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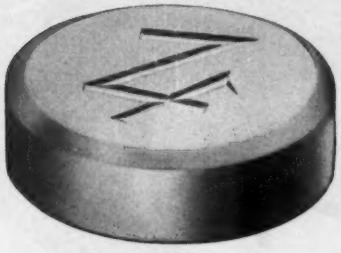

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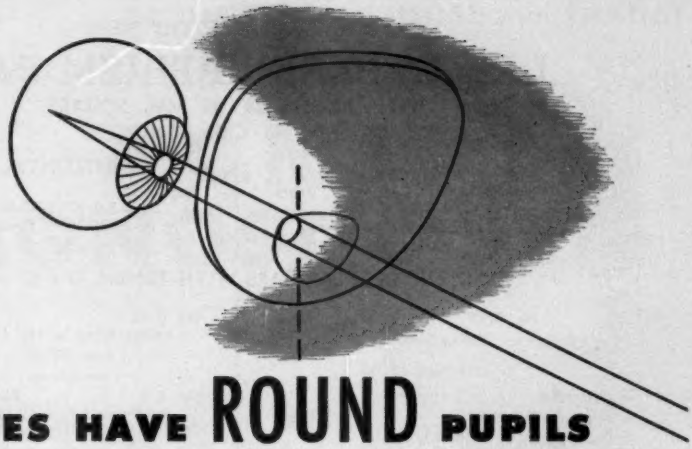

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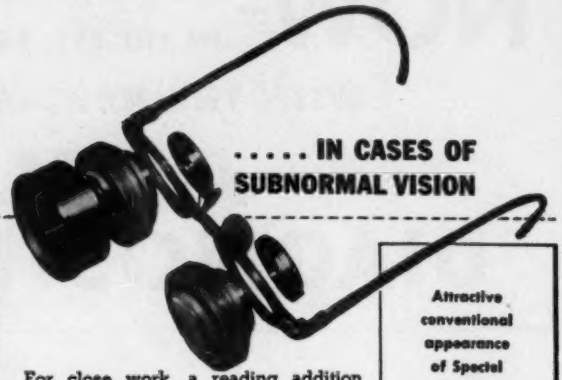

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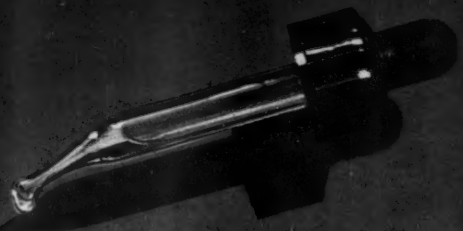

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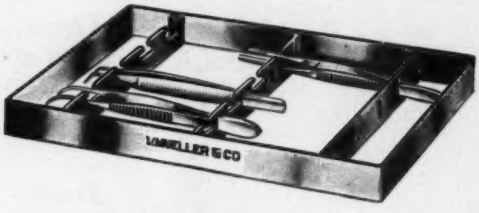

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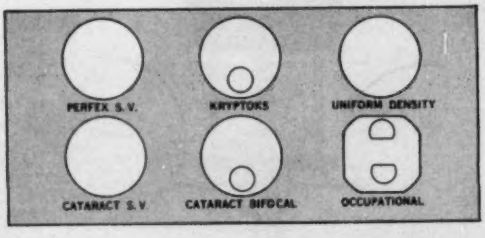

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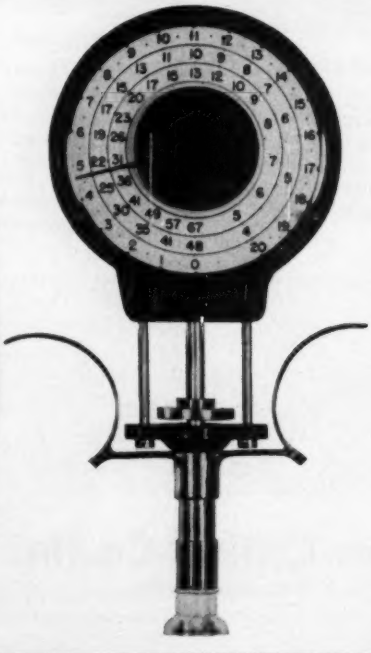

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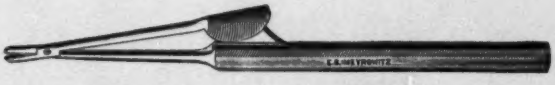

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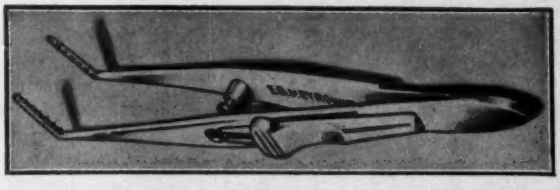

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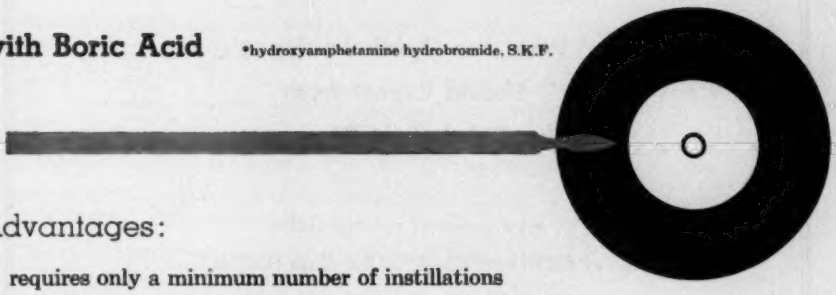

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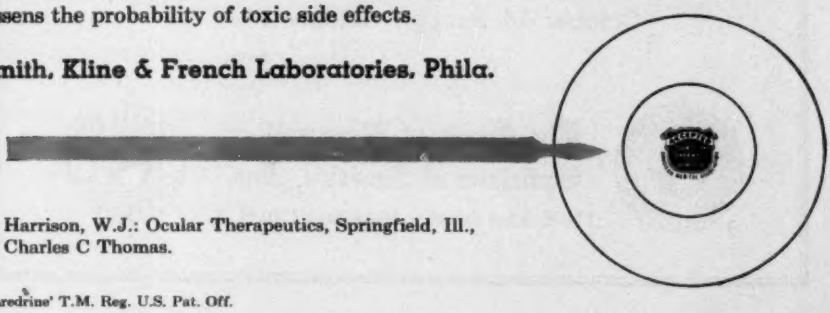

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TERRAMYCIN IN THE TREATMENT OF TRACHOMA

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IN A PREVIOUS report by two of us,[1] preliminary information on the use of terramycin in the treatment of trachoma was given, and the high response of trachoma to this antibiotic was described. In the present study, approximately 700 patients with trachoma, in various stages, were treated with this antibiotic, under an assignment planned by one of us (Y. M.)

Five forms of crystalline terramycin hydrochloride[2] were utilized in the present study: crystalline terramycin hydrochloride capsules, for oral administration; crystalline terramycin hydrochloride ophthalmic ointment (0.1%); crystalline terramycin hydrochloride ophthalmic solution (0.5%), and a 0.5 and a 1.0% terramycin hydrochloride ointment compounded in a petrolatum base.

ACUTE TRACHOMA

Thirty-eight patients with acute trachoma have been treated to date, including those reported on previously. In 11 of these 38 patients the trachoma was of two-to-seven-days' duration. Six of these 11 patients were treated with 0.5% terramycin hydrochloride ointment four to nine times a day; 3 patients, with 0.1% terramycin hydrochloride ophthalmic ointment four times a day, and 2 patients, with 0.5% terramycin hydrochloride solution four to six times a day. All patients were cured in the course of seven to 10 days, without exception.

In the remaining 27 patients, the trachoma was of three-to-eight-weeks' duration. They were divided into four groups and treated as follows (Table 1):

Systemic Administration.—For two patients, 2 gm. of terramycin hydrochloride was divided daily into two doses, to be administered orally for 14 days. The inclusion bodies disappeared after two days. Clinical symptoms, however, responded rather slowly, and two-weeks' treatment was required for a reduction of acute signs. The follicles persisted to a considerable degree until the end of the second week. When ointment therapy (0.1% opthalmic ointment, 12 times a day) was substituted for the systemic treatment, the condition began to improve

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1. Mitsui, Y., and Tanaka, C.: Terramycin, Aureomycin and Chloramphenicol in Treatment of Trachoma, Antibiotics & Chemother. **1**:146-157 (May) 1951.

2. Supplied by Dr. R. A. Patelski, Chas. Pfizer & Co., Inc., Brooklyn.

rapidly and was cured two to three weeks later. A slight gastrointestinal disturbance was observed in one patient during the systemic administration. Comparison of this result with the results to be discussed below indicates that a systemic administration of terramycin may be less effective than its topical use in acute trachoma.

Topical Treatment.—Ointment, 0.5%: To 11 patients, a 0.5% terramycin ointment was given three to seven times daily. Inclusions disappeared within one to five days. A perfect cure, judged by the clinical appearance, resulted after a course of two to three weeks.

To two other patients the ointment was given every hour during the day and less frequently during the night. The response of acute symptoms was more rapid than in the patients mentioned above, but follicular hypertrophy did not respond more rapidly. The total course of the disease, therefore, was not obviously shortened.

TABLE 1.—*Comparison of Effects of Terramycin by Various Methods of Administration on Acute Trachoma of Three- to Eight-Weeks' Duration*

Regimen	Times Daily	No. of Cases	Post-Therapy Period Until Disappearance of Clinical Signs: Inclusions (Days)	Discharge (Days)	Hyperemia (Days)	Follicles (Wk.)	Result
Capsules	2*	2	2	7-10	14	4-5†	Unsatisfactory
0.5% ointment	3-7	11‡	1-5	2-10	5-14	2-3	Satisfactory
	12-24	2	2	2-3	3-4	2-3	Satisfactory
0.1% ointment	2	2	>10	>10	>10	4§	Unsatisfactory
	4	6‡	1-3	2-5	5-12	2-3	Satisfactory
	12-24	2	2	2-3	3-5	2-2.5	Satisfactory
0.5% solution	4	3	2-3	3-6	4-8	2.5-3	Satisfactory
	12-24	2	2	3	3-4	2-3	Satisfactory

* Each 1 gm. for 14 days.
† After the 14th day ointment therapy was substituted.
‡ In 3 cases the right eye was treated with 0.5% ointment and the left eye with 0.1% ointment.
§ After the 10th day eye drops were used 12 times a day.

Ointment 0.1%: With two patients the ointment was applied twice daily. Improvement in clinical appearance was slow. Inclusions were present even after 10 days. Eye drops were used thereafter, 12 times a day. The response was then prompt and rapid, and a perfect cure resulted about 20 days thereafter. Application of 0.1% ointment twice daily seems unsatisfactory for acute trachoma.

Six patients were then treated with 0.1% terramycin ointment four times daily. The response was rather rapid and satisfactory, and a perfect cure resulted within two to three weeks, as judged from the clinical appearance.

Two other patients received the ointment every hour during the day and less frequently during the night. Acute symptoms responded far more rapidly, but the whole course was not obviously shortened.

Solution, 0.5%: With three patients the solution was used four times a day. The response was rather rapid and satisfactory; a perfect cure, as judged from the clinical appearance, resulted within 2.5 to three weeks.

To two other patients the solution was administered every hour during the day and less frequently during the night. The response of acute symptoms was then far more prompt, but the course of the disease did not become obviously shorter.

Relapses.—In five instances at least, in which the treatment was stopped immediately after the clinical cure was effected, relapses occurred.

Seven patients who had been treated for at least two weeks after clinical cure were examined five to six months after the completion of treatment. It was revealed that in these patients the cure was complete and permanent.

In summary, it may be said that 0.1 or 0.5% of terramycin hydrochloride ointment, as well as 0.5% terramycin hydrochloride solution, applied more than four times a day is satisfactory in the treatment of acute trachoma, but a too early discontinuation of treatment often will be followed by an inflammatory relapse. Treatment for about two weeks after a clinical cure will result in permanent cure, without a failure.

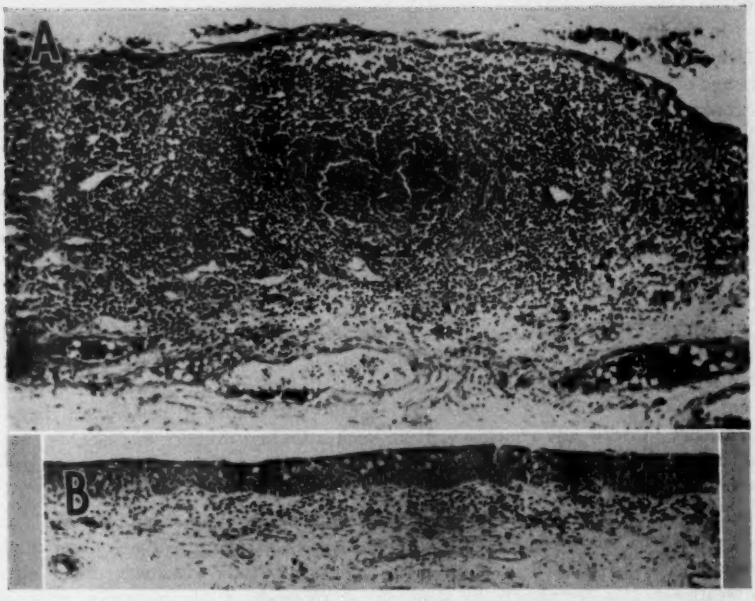

Fig. 1.—Histopathological changes of the earliest stage of trachoma, of nine-days' duration in the right eye, and of five-days' duration in the left eye.

A indicates the fornix conjunctivae of the right eye before treatment; *B*, the fornix conjunctivae of the left eye after 10-days' treatment with 0.1 % terramycin ointment four times daily. Clinical signs before the treatment were a little more pronounced in the right eye.

However, inflammatory relapses responded promptly to re-treatment with terramycin, and cure was effected in the course of three to four weeks. Therefore there was no clinical evidence of the development of resistance of the trachoma virus to terramycin in these cases.

Relapse after terramycin treatment was insidious. We reported a relapse in two of eight cases of acute trachoma in an aureomycin series.[1] The relapse in these cases appeared rather acute. Our experience indicates, further, that with aureomycin, inclusion bodies at first lose their blue tint and then gradually disappear, whereas with terramycin they lose their red tint, then rather suddenly disappear. There may be some difference in the mode of action of these two antibiotics.

According to a histochemical study on inclusion bodies by Nakashima,[3] terramycin seems to affect the desoxyribonucleic-acid (DNA) system, while aureomycin seems to affect the ribonucleic-acid (RNA) system.

CHRONIC TRACHOMA

In preliminary observations in a small series of cases of chronic trachoma,[1] it was suggested that in chronic trachoma (1) treatment for two or three months may be required for a perfect cure; (2) treatment for one month or less may be followed by a cure during a one-to-two-month follow-up period, and (3) a combination of surgery and chemotherapy may shorten the course of the disease considerably.

In the present study, therefore, approximately 640 patients with typical chronic trachoma were selected. They were divided into several groups, and the optimum dosage of terramycin and the effect of combined surgical and antibiotic treatment of chronic trachoma were studied.

Material.—About 640 patients with typical chronic trachoma were selected from among about 10,000 students at seven primary and four junior high schools, where the incidence of trachoma was more than 15%. Of these 640 cases, 190 were severe, 270 were moderately severe, and 180 were mild. Ten patients from each of the three groups—with severe, moderately severe, and mild disease respectively—were selected at random prior to treatment and examined for inclusion bodies. It was then revealed that nine of 10 patients with severe, five of 10 patients with moderately severe, and two of 10 patients with mild chronic trachoma had inclusion bodies.

Systemic Administration.—To two patients, 2 gm. of terramycin hydrochloride, divided into two doses, was administered orally every day for 14 days. Reduction of discharge and hyperemia began during the treatment, but only slight improvement in follicle formation resulted during a 10-wk. follow-up period. It may be premature to draw any conclusion from such a limited number of cases, but it is our impression that systemic administration alone is less effective than appropriately performed topical treatment.

Topical Treatment.—About 550 patients were divided into several groups. Care was taken to make a uniform distribution of cases in relation to the severity of the symptoms. As indicated in Table 2, a comparative study was made of the effect of treatment with terramycin by a number of methods of administration, differing with respect to (1) form and concentration of terramycin, (2) frequency of application, and (3) duration of treatment.

The first sign of improvement, such as reduction of hyperemia and discharge, was apparent in the majority of cases after a course of treatment of one to two weeks. During the period of two-to-six-weeks' treatment, however, a complete cure was observed in only about 20% of the patients, even in the group treated for six weeks, though considerable improvement was usual. In the group treated for two weeks complete cure during the period of treatment was only the exception.

Follow-up observation of the course of the disease after completion of the treatment, however, revealed that in most cases the symptoms continued to decrease spontaneously after the treatments were discontinued, until a perfect cure resulted during a follow-up period of one to two months. But in some cases the reduction of symptoms ceased on discontinuation of the treatment; in others the inflammation even began to increase. Thus, in the groups treated with 0.5% ointment three

3. Nakashima, A.: Personal communication to the authors.

times a day for more than two weeks, with 0.5% solution three times a day for more than four weeks, and with 0.1% ointment three times a day for six weeks, cures were observed in about 80% of the patients in a course of 12 wk. after the beginning of treatment. In other groups, the cure was complete in 40 to 70% of the patients (Table 2).

TABLE 2.—*Terramycin in Treatment of Chronic Trachoma*

Regimen	Duration of Treatment (Wk.)	No. of Patients	After 12 Wk.: No. of Patients		
			Cured	Improved	Not Improved*
Systemic, 2 gm. daily	2	2	0 (0%)	2	0
0.5% ointment, thrice daily	2	46	39 (85%)	5	2
	4	43	35 (81%)	4	4
	6	15	12 (80%)	1	2
Total	..	104	86 (83%)	10	8
0.5% ointment, once daily	2	37	23 (62%)	8	6
	4	38	25 (66%)	8	5
	6	19	13 (68%)	5	1
Total	..	94	61 (65%)	21	12
0.1% ointment, thrice daily	2	53	29 (55%)	12	12
	4	50	30 (60%)	8	12
	6	16	13 (81%)	1	2
Total	..	119	72 (61%)	21	26
0.1% ointment, once daily	2	17	7 (41%)	2	8
	4	33	21 (64%)	5	7
	6	26	17 (65%)	4	5
Total	..	76	45 (59%)	11	20
0.5% solution, thrice daily	2	55	37 (67%)	12	6
	4	40	32 (80%)	5	3
	6	29	23 (79%)	5	1
Total	..	124	92 (74%)	22	10
Total	..	519	356 (69%)	87	76

* Including patients whose symptoms increased in severity.

TABLE 3.—*Effect of Combined Surgical and Terramycin Treatment in Patients with Chronic Trachoma*

Regimen*	No. of Eyes	After Treatment: Total No. of Eyes						
		Cured Within (Wk.)					Improved	Not Improved
		4	6	8	10	12		
Severe Trachoma								
Surgery and terramycin	51	11	32	40	40	42	9	0
Terramycin	51	0	0	15	28	35	14	2
Moderate Trachoma								
Surgery and terramycin	38	26	34	36	36	36	2	0
Terramycin	38	2	8	11	25	30	5	3

* The right eye was treated with combined surgical and terramycin treatment, and the left eye, with terramycin only; surgically, the follicle was expressed prior to terramycin treatment, and, as terramycin treatment, 0.5% ointment or solution was used three to six times a day for four to six weeks.

The question arises whether or not a severe form of trachoma is difficult to cure. Of 517 patients who had been treated for more than two weeks, 123 had severe, 224 moderately severe, and 170 mild, trachoma. Of 123 patients with severe trachoma, 71 (58%) were cured; 42 were improved, and 10 were not improved. Of 224 patients with moderately severe disease, 157 (70%) were cured; 39 were improved, and 28 were not improved. Of 170 patients with mild trachoma, 128 (75%) were cured; 4 were improved, and 38 were not improved.

The problem of the minimum dose for most terramycin-sensitive patients is a matter of concern. There were 34 patients who, owing to circumstances, were treated irregularly and for less than a week. Examination after 12 wk. revealed that of these 34 patients 10 (29%) were cured, 3 were improved, and 21 were not improved.

It may be said, therefore, that, for chronic trachoma the optimum dosage varies greatly from case to case. An increase in dosage is, of course, beneficial in general. But a severe condition is not always difficult to cure; on the contrary, a mild form is not always easy to cure.

Ointments and Solution.—The results summarized in Table 2 indicate that the effect of 0.5% ointment may be slightly superior to that of the 0.5% solution, pro-

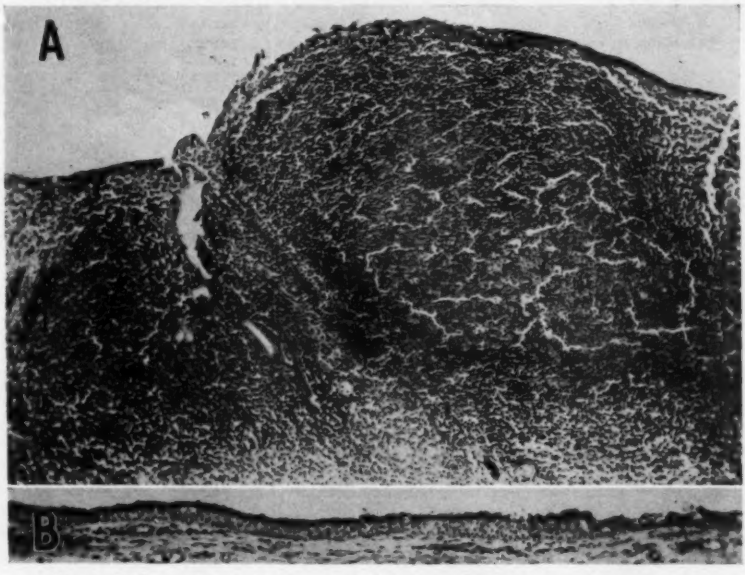

Fig. 2.—Histopathological changes of chronic trachoma in an advanced stage.
A indicates the fornix conjunctivae of the left eye before treatment; *B*, the fornix conjunctivae of the right eye after four-weeks' treatment with a combination of surgical and terramycin therapy (0.5% ointment, three times daily).
Clinical findings before the treatment were the same in two eyes.

vided that the two forms are used similarly. A 0.1% ointment used three times a day may be more effective than a 0.5% ointment used once a day.

Provocation of Symptoms.—In 7 of 517 patients treated regularly, an increase in severity of symptoms occurred during treatment. These patients were mainly in the group receiving 0.1% ointment once a day. Follow-up observation of the further course of the disease revealed that in two of these seven patients the symptoms subsided again. But in the remaining five patients severe symptoms persisted until the end of the 12th week. Examination of scrapings revealed the presence of inclusion bodies. The question whether or not the increase of symptoms in these cases was really provoked by terramycin is not yet answered. But there may be such an occurrence in exceptional instances.

Effect of Combined Surgical and Terramycin Therapy.—Fifty-one patients with severe and 38 patients with moderately severe disease were selected as the subjects of this study. The right eye was operated on prior to terramycin treatment, and both eyes were treated with terramycin similarly thereafter. Surgically, an expression of the follicle was performed, and, as terramycin treatment, a 0.5% ointment or solution was used three to six times daily for four to six weeks. As indicated in Table 3, in the series of severely affected patients, the combined treatment resulted in a cure in 82% of the eyes, whereas the simple method resulted in a cure in 70% of the eyes; the average time until cure was effected was 6.1 wk. with the combined method and 9.5 wk. with the simple method. In the series of patients with moderately severe disease, the cure was perfect in 95% of the eyes with the combined method, and in 79% of the eyes with the simple method; the average time until cure was 4.7 wk. with the former and 8.9 wk. with the latter. No doubt can remain, therefore, that the combined surgical treatment and chemotherapy not only shortened the course of the disease considerably but produced a cure in an increased number of cases.

Second Course of Treatment.—To 56 of 87 patients who were, though improved, not cured by the first course of treatment, a second course of therapy was given.

TABLE 4.—*Results of the Second Course of Treatment in Chronic Trachoma*

Regimen	No. of Patients	Post-Therapy: No. of Patients			
		Cured Within		Improved	Not Improved
		6 Wk.	12 Wk.		
Patients Improved But Not Cured by First Course					
0.5% ointment or solution	56	49	54 (96%)	2	0
Patients Not Improved by First Course					
0.5% ointment	20	6	14 (70%)	5	1
1.0% ointment	20	5	16 (80%)	4	0
Combined surgery and terramycin	20	16	19 (95%)	1	0

A 0.5% ointment or solution was used three to six times a day for four to six weeks. By the end of the course of treatments 49 patients (87%) were cured, and seven were considerably improved. By the end of the 12th week only 2 patients had persistence of slight symptoms. Thus, 54 of the 56, or 96%, of the patients were cured in the course of 12 wk. (Table 4).

In 60 of 76 patients who were not improved by the first course of treatments, including five with an increase in symptoms, a study of the results of the second course of treatments was performed as follows: The 60 patients were divided into three groups of 20 patients each. The first group was treated with 0.5% ointment; the second group, with 1.0% ointment, and the third group, with combined operation and application of 1.0% ointment. In each case, ointment was used three times daily for six weeks. Follow-up observation on the course of the disease for 12 wk. revealed that in these patients the combined surgical and terramycin therapy resulted in a cure in 95% whereas simple methods resulted in cure in 70 to 80% (Table 4). A 1.0% ointment seemed a little more effective than a 0.5% ointment.

COMMENT

Chronic trachoma responds fairly well to terramycin. The best dosage of terramycin in the treatment of chronic trachoma cannot, however, be determined for all cases. In some cases, a small dosage, such as use of a 0.1% ointment once a day

for less than a week, is enough for a complete cure, whereas in some others a greater dosage, such as 0.5% ointment given three times a day for six weeks, is nevertheless unsatisfactory. Severe trachoma is not always difficult to cure, and the mild form is not always easily cured. In general, however, one of the following methods of administration will result in a complete cure in about 80% of cases: use of a 0.5% ointment three times a day for more than two weeks; use of a 0.5% solution three times a day for more than four weeks, and use of a 0.1% ointment three times a day for more than six weeks. It must be borne in mind that in most cases a cure does not occur during the treatment, but it does occur during a follow-up period of one to two months.

Terramycin treatment combined with surgery may not only shorten the course of the disease considerably but also decrease the number of cases in which another course of therapy will be required. Of a number of surgical methods, it is our impression that expression of the follicles is the best.

In cases in which a first course of treatments has failed to cure, a second course of treatment is recommended. A second course of treatments is, of course, not indicated immediately after the first course, but at least one or two months thereafter. In cases in which improvement, but not cure, followed the first treatments, the same technique as that used in the first course may be satisfactory. In cases in which no improvement at all followed the first treatments, however, a combination of surgical intervention and chemotherapy is indicated. Thus, about 95% of patients with chronic trachoma will be cured in the course of five to six months. The problem of whether or not there are exceptional cases in which the trachoma will not respond to terramycin at all, as well as the problem of development of resistance of the trachoma virus to terramycin, are to be studied in the future.

An ointment seems a little more effective than a solution of the same concentration when they are similarly used. This may be because of the longer retention of an ointment in the conjunctival sac.

In general, it seems more beneficial to administer the same amount of terramycin in frequent doses of low concentration than in fewer doses of strong concentration.

Use of too small a dosage, such as application of a 0.1% ointment once a day, might in exceptional instances provoke inflammatory symptoms. In such cases a prompt increase in the dose seems advisable.

Systemic administration seems to have no advantage over topical application—the former may even seem inferior to the latter. This may be because of the nature of trachoma as a surface disease of the conjunctiva. The causal agent of trachoma exists in the epithelial layers, and subepithelial changes are secondary to changes there.[4] Disappearance of the causal agent and disappearance of subepithelial changes are two different things. In the acute stage of trachoma, subepithelial changes are little resistant, while in chronic stages they are highly resistant. In acute trachoma, therefore, secondary changes may be absorbed prior to eradication of the causal agent, whereas in chronic trachoma the secondary changes are likely, in general, to persist for a certain period after eradication of the

4. Michail, D., and Vancea, P.: Quelques faits expérimentaux dans le trachome, Rev. internat. trachome **9**:33-36, 1932; cited by Thygeson, P.: Trachoma and Inclusion Conjunctivitis: Viral and Rickettsial Infection of Man, Philadelphia, J. B. Lippincott Company, 1948, pp. 358-369. Okamura, K., and Mitsui, Y.: Contagious Trachoma Virus in Subconjunctival Tissue, Acta Soc. Ophth. Jap. **43**:95-101 (Jan.) 1939.

cause. This may be the reason that in cases of acute trachoma prolonged treatment after a clinical cure is required for a permanent cure, whereas in chronic cases early completion of treatment is often followed by cure during a certain follow-up period.

The problem as to why a higher concentration of terramycin is required in the chronic stage than in the acute stage, and the problem of the absence of a uniform response in the chronic stage, are discussed. In the acute stage the virus is in the most active state, while in the chronic stage the virus becomes rather static. According to Tsutsui,[5] the response of certain bacteria to terramycin parallels the activity of the organisms. In Staphylococcus in the static state, for instance, more than ten times as great a concentration of terramycin is required to interfere with respiration (oxygen consumption, measured by Warburg's method) than is needed in the state of active multiplication. This may answer the first question,

TABLE 5.—*Effect of Terramycin in the Treatment of Late-Stage Trachoma with Corneal Invasions*

Regimen	Times Daily	No. of Patients	Post-Therapy Period Until Disappearance of Clinical Signs, Days
Ordinary Acute Pannus			
0.5% ointment	1	3	2-5
	3	4	1-3
0.1% ointment	1	4	2-6
	3	10	1-6
0.5% solution	3-6	4	2-5
	12-24	3	2-6
Capsules	2*	2	4-5
Pannus Crassus			
0.1% ointment	12-24	2	12-15
0.5% solution	12-24	2	10-14

* Systemically, each 1 gm. for 3 days.

provided that the matter is much the same with viruses. The second question may be answered by the variable activity of the trachoma virus in the chronic stage.

LATE-STAGE TRACHOMA WITH CORNEAL INVASIONS

Thirty-four patients with the late stage of trachoma, with acute pannus and/or ulceration of the cornea, were treated with terramycin (Table 5). Thirty of these 34 patients had ordinary acute pannus and/or ulceration of the cornea. Two of them received 2 gm. of terramycin daily, divided into two doses and administered orally, for three days. There was a prompt response, and acute symptoms and corneal ulcers disappeared in the course of four to five days. With 28 other patients, ointments or solution were used as follows: Three were given 0.5% ointment once daily; 4, the same ointment three to four times a day; 4, 0.1% ointment once a day; 10, the same ointment three times a day; 4, 0.5% solution three to six times a day, and the last three patients, the same solution 12 to 24 times a day. The response was prompt and satisfactory in all patients. An improvement in subjective symptoms, such as pain and photophobia, was apparent within 12 to 24 hr., and acute symptoms and corneal ulceration were cured in the course of one to six days. In most cases the cure was followed by considerable

5. Tsutsi, J.: A Study on the Mode of Action of Aureomycin and Terramycin, Folia ophth. jap. **2**:164-166 (March) 1951.

improvement of the visual acuity during a follow-up period of a few weeks. In one case, in which the treatment had been stopped immediately after the cure of acute conditions, a relapse occurred, which, however, responded promptly to re-treatment with terramycin. A small dose, for instance, 0.1% ointment given once daily, may be satisfactory in most cases of the acute condition, but prolonged treatment may be advisable for prevention of relapse.

In the remaining four patients, the pannus was of a hypertrophic nature—the so-called pannus crassus. In these patients, 0.1% ointment, or 0.5% solution, was used 12 to 24 times a day. The response was rather slow, and, though improvement was apparent within 24 hr., 10 to 15 days was required for a full cure.

CONCLUSIONS

On the basis of a study of treatment with terramycin in about 700 cases of trachoma the following conclusions are reached:

Early trachoma of less than two-months' duration will respond promptly and well to terramycin without exception. A 0.1% terramycin hydrochloride ointment, as well as a 0.5% solution of the drug applied more than four times a day, will produce a clinical cure in the course of a few weeks. Too early discontinuation of treatment, however, is often followed by an inflammatory relapse. Treatment for two weeks after a clinical cure will be satisfactory for a permanent cure.

Chronic trachoma of several years' duration will respond fairly well to terramycin, but the best dosage of terramycin cannot be determined for all cases. A 0.5% ointment applied three times a day for four weeks, however, will be followed by cure in about 80% of cases, occurring during a one-to-two-month follow-up period after the completion of treatment. A combination of surgical and terramycin therapy will give far better results. In cases in which the first course of treatments fails to cure, a second course of treatments is to be given, with an increased dose of terramycin or with surgical therapy combined with topical use of the drug. A third course of treatment will not be required in more than 5% of cases.

Acute pannus and ulceration of the cornea will uniformly respond promptly to terramycin treatment. Application of a small dose will produce a cure within several days. In pannus crassus, however, two weeks' treatment will be required before healing is complete, even with an increased dose.

In all these conditions, systemic administration alone seems to have little advantage over topical application. An ointment seems to be slightly more effective than a solution.

NOTE: Since this paper was submitted for publication, the following results have been obtained:

1. During a nine-month period, among the patients with chronic trachoma cured by the first course of treatment, and actual relapse was observed in only four of the 243 patients examined.

2. A combination of terramycin ointment (0.5 or 1.0%, applied three times a day for six to 10 weeks) and sulfadiazine (1.5 gm. daily given by the oral route for the first 10 days) resulted in cure in 15 of the 15 patients with chronic trachoma treated.

3. A terramycin (0.5%)-hyaluronidase (1 turbidity-reducing unit [TRU] per gram) ointment applied three times a day for six to eight weeks resulted in cure in 10 of the 11 patients with chronic trachoma treated.

HORIZONTAL SQUINT WITH SECONDARY VERTICAL DEVIATIONS

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IN NORMAL eyes with good fusion heterophoria is commoner than orthophoria. When the eyes are dissociated by the cover test, fusion is interrupted for a short time, and the eye under cover will move into the dissociated position. The dissociated position is influenced by the anatomical factors determining the position of the eyes, such as orbital assymmetry, size of eyeballs, interpupillary distance, check ligaments, and insertion of muscles. A good discussion of these factors can be found in "Worth's Squint" by Chavasse.[1] The dissociated position is also influenced by innervational factors, such as convergent and divergent impulses and accommodative, vestibular, cerebellar, and other reflexes.

When normal eyes are dissociated for a period of time, as in the Marlow prolonged-occlusion test, vertical deviations are found to be just as prevalent as lateral deviations. Marlow [2] was a pioneer in working for a better understanding of the dissociated position of the eyes. With his well-known prolonged-occlusion test, he measured the positions the eyes assumed after they were dissociated for periods varying from one day to three weeks. Of 700 subjects in whom one eye was occluded from one to 20 days, 84% had a hyperphoria after occlusion. Of 82 subjects who had occlusion of first one eye and then the other, 98% had a hyperphoria. These findings of a high incidence of hyperphoria in eyes after prolonged occlusion have been corroborated by Fink,[2a] Clark,[3] Swab,[4] and many others. Marlow [5] himself, in one of his last articles, appreciated his fundamental discovery that 98% of eyes that are dissociated by prolonged occlusion show a hyperphoria, when he said:

> The phorias and ductions can be accounted for on an evolutionary basis. The idea that many of the vertical deviations can be attributed to a paresis is without sufficient justification. They occur in about the same frequency as, and often in association with, lateral deviations.

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1. Chavasse, B.: Worth's Squint, Ed. 7, London, P. Blakiston's & Sons, 1939.

2. Marlow, F. W.: Recent Observations in the Prolonged Occlusion Test, Am. J. Ophth. **16**:519, 1933; The Relative Position of Rest of the Eyes and the Prolonged Occlusion Test, Philadelphia, F. A. Davis Company, 1924.

2a. Fink, W. H.: Significance of Ocular Muscle Imbalance, Am. J. Ophth. **10**:168, 1927.

3. Clark, C. P.: Diagnosis of Lateral and Vertical Deviations of the Eyes by Use of Prolonged Monocular Occlusion, Am. J. Ophth. **10**:574, 1927.

4. Swab, C. M.: Latent Heterophoria After Prolonged Monocular Occlusion, Am. J. Ophth. **13**:1054, 1930.

5. Marlow, F. W.: A Tentative Interpretation of the Findings of the Prolonged Occlusion Test on an Evolutionary Basis, Arch. Ophth. **19**:194 (Feb.) 1938.

He believed that orthophoria was the culmination of the evolutionary process and that the hyperphorias brought out by the prolonged-occlusion test might be deviations found in the prebinocular vision period in the development of man.

Bielschowsky,[6] working with eyes dissociated for a longer period than in the Marlow occlusion test, also found a high incidence of vertical deviation. Of a series of 289 patients with dissociated eyes due to disease or injury to one eye, with loss of binocular vision, he found a vertical component in 120, or 41%. Most of these subjects had an alternating hypertropia combined with a horizontal squint. Bielschowsky believed that these vertical deviations were due, not to paretic vertical muscles, but to innervational factors acting on eyes that were dissociated.

Chavasse [1] was acutely aware of the idea that vertical deviations could be secondary to horizontal squint. He stated: "The cyclovertical deviations have constituted . . . one of the most baffling of all the problems which present themselves in the kaleidoscopic panorama of strabismus." He went on to say that it is important to make the distinction between "a primary cyclovertical deviation and one which is secondary to, or implicit in, a horizontal deviation." This viewpoint is supported by the finding of a high incidence of vertical deviations associated with horizontal concomitant squint by many authors. Pugh,[7] on analyzing 214 cases of horizontal squint subject to operation, found a vertical component along with the horizontal squint in 42%. Bielschowsky [8] found that 45% of horizontal deviations had a vertical component. Dunnington and Regan [9] observed a vertical component in 50% of 79 cases of concomitant convergent strabismus. Enos,[10] in a study of 176 cases of heterotropia, found a vertical component in 60%. White and Brown [11] noted a vertical component in about one-third of their cases of lateral deviation.

In a series of 615 patients with horizontal strabismus whose deviation we measured by the prism-screen test at the Illinois Eye and Ear Infirmary motility clinic, 431, or 79%, showed a vertical component along with the horizontal deviation. Of these 431 cases, 198, or 43%, showed a bilateral vertical component, that is, one type of vertical deviation on gaze to the right and the opposite vertical deviation on gaze to the left. These vertical deviations were hypertropias in some subjects and hyperphorias in others, and many patients had hyperphorias which became hypertropias at times. It is the purpose of this paper to report the methods of examination and findings in these cases of predominantly horizontal squint with smaller degrees of vertical deviations. The importance of observing the position of the eyes when looking straight up and straight down will be demonstrated, and a new classification of these types of strabismus will be presented.

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6. Bielschowsky, A.: Die einseitigen und gegensinnigen (dissoziierten) Vertikalbewegungen der Augen, von Graefes Arch. Ophth. **125**:493, 1931.

7. Pugh, M.: A Classification of Cases of Concomitant Strabismus Based on the Aetiological Factor, Brit. J. Ophth. **18**:446, 1934.

8. Bielschowsky, A.: Lectures on Motor Anomalies, Am. J. Ophth. **21**:843, 1938.

9. Dunnington, J. H., and Regan, E. F.: Factors Influencing the Postoperative Results in Concomitant Convergent Strabismus, Arch. Ophth. **44**:813 (Dec.) 1950.

10. Enos, M. V.: Suppression Versus Amblyopia, Am. J. Ophth. **27**:1266, 1944.

11. White, J. W., and Brown, H. W.: Occurrence of Vertical Anomalies Associated with Convergent and Divergent Anomalies, Arch. Ophth. **21**:999 (June) 1939.

METHODS OF EXAMINATION

It is necessary to describe the method of examination in some detail in order to show how the variations in the vertical deviation found with horizontal squint are best observed. An effort was made to determine what the patient's eyes did under normal, everyday conditions. Certainly, the physician's examining room or the hospital admitting room is not the best place to determine this. A reliable history from the mother helps greatly in understanding what the patient's eyes do under normal conditions, that is, when he is absorbed in play or work and not thinking of his eyes.

Questions were asked whether the eyes were ever straight or what per cent of the time they were straight, which eye deviated, how it deviated, and under what conditions. The mother's observations on the child's eyes at home were verified by having her describe how they looked to her in the clinic, e. g., which eye was crossed and to what degree, and whether the eyes were straighter at home or at the clinic. To gain the child's confidence, a sufficient number of visits were made that he was no longer frightened and apprehensive. An occasional sweet helped a great deal.

The first step in the examination was casual inspection of the eyes when the child was unaware of it. Then the patient was instructed to look at an object across the street or at a letter or light at 20 feet (6 m.), at a word in a book, or at a small toy or picture. The patient then fixed a muscle light, which for small children was the light in the nose of "Rudolph, the Red-Nosed Reindeer," and the angle of squint was determined by the Hirschberg method. Next, versions were observed in following a light straight up, straight down, to the right, to the left, and in the oblique positions of gaze. Convergence was also tested.

While studying the position of the eyes in these versional movements, we noticed to our surprise that there were tremendous changes in the horizontal angle of squint in certain cases both of esotropia and of exotropia as the patient's eyes followed a light straight up and down. Also, the versions in lateral gaze often revealed a change in the vertical relation of the eyes, especially an elevation or depression of the adducted eye. The term "elevation or depression in adduction" was used to describe this condition. The patients who showed the greatest change in their horizontal deviation as the versions were taken in a vertical direction also showed a great amount of change in their vertical deviation when the versions were taken horizontally. After the versions were determined, the patient's eyes were photographed for distance and for near, looking up, down, right, and left, and to show the convergence near point. In cases in which there was a primary vertical deviation photographs were taken in all the positions of gaze.

The prism-screen, or cover, test was then performed in all the directions of gaze outlined above for versions, to obtain quantitative measurements. Since many of these patients showed a vertical deviation of one type, such as a right hypertropia, on looking to the left, and a vertical deviation of the opposite type, such as a left hypertropia, on looking to the right, the problem was what kind of vertical deviation these patients had in looking straight ahead. Instead of rapidly covering each eye in turn, we allowed one eye to fix the light steadily for one-half minute before shifting the cover to the other eye. This was called the "delayed cover test." The majority of these subjects showed the condition that Bielschowsky called dissociated vertical divergence with horizontal squint, of the following types: (1) bilateral alternating hypertropia, each eye being up under cover; (2) bilateral alternating hypotropia, each eye being down under cover; (3) one eye being up under cover, the other eye remaining horizontal under cover; (4) one eye being down under cover, the other remaining horizontal under cover.

A few other patients showed concomitant vertical deviations, with one eye up and the other eye down under cover. Many of these patients also showed bilateral wheel rotation in; that is, when the cover was removed from the nonfixing eye, the upper end of the vertical meridian of the cornea rotated nasally as the eye moved to fix. Occlusion, or latent, nystagmus was noticed in some cases; that is, with both eyes open and fixing a light, there was no nystagmus, but when one eye was covered, the fixing eye had nystagmus along with the occluded eye.

All the patients had had a previous atropine refraction; visual acuity was determined if possible, and fusion was tested on the major amblyoscope.

PRESENTATION OF CASES

After a careful study of the versional movement of our patients, along with the quantitative study of these movements with the prism-screen test in all positions of gaze, the combination of horizontal squint with a vertical component was found more frequently as we looked for it. These patients were then studied to see whether the combination of vertical and horizontal deviations could be classified. More than 50% of the patients with this combination could be classified under four clinical entities:

Group I: Esotropia with bilateral elevation in adduction (200 cases)
Group II: Esotropia with bilateral depression in adduction (66 cases)
Group III: Exotropia with bilateral elevation in adduction (74 cases)
Group IV: Exotropia with bilateral depression in adduction (30 cases)

TABLE 1.—*Esotropia (in Degrees) with Bilateral Elevation in Adduction*

	Esotropia Far	Esotropia Near	Esotropia Up	Esotropia Down	LH(△) on Gaze to Right*	RH(△) on Gaze to Left*	NPC,† Mm.	Grade Fusion	Delayed Cover Test‡
T. S.	35 LH 2	44 LH 2	18 LH 3	65	GU 7	GU 2	30	1st	AHo
J. K.	54 RH 18	67 RH 10	60	90	GU 5	GU 20	25	None	AH
R. Z.	60 RH 15	67 RH 14	28 RH 16	60 RH 5	GU 7	GU 20	40	2d	AH
R. B.	53 RH 14	56 RH 8	52	65	GU 5	GU 15	40	None	NT
W. G.	54 RH 12	60 RH 10	36 RH 5	63	GU 4	GU 7	25	2d	AHo
G. B.	50 LH 5	54 LH 5	44	70	GU 8	GU 3	20	1st	NT
L. H.	30	35 RH 4	20 LH 8	30 RH 12	GU 8	GU 12	25	3d	LHo R—
S. R.	14 RH 9	26 RH 5	9 RH 6	30	GU 7	GU 25	25	..	RH L—
P. D.	42 RH 3	47 RH 2	34 RH 2	60	GU 2	GU 5	25	2d	AH
W. B.	49 RH 4	57 RH 2	10 RH 3	70	GU 2	GU 8	30	2d	NT
S. S.	30 LH 4	36	16	58	GU 10	GU 12	30	1st	AH
L. K.	20 LH 2	25 LH 2	24 LH 2	28	GU 2	GU 6	45	2d	NT
M. K.	42 LH 20	48 LH 16	27 LH 8	38 LH 12	GU 22	GU 20	40	..	AH WR
P. R.	40	50	45 RH 7	50 RH 7	GU 5	GU 7	25	..	AH
M. P.	22 LH 2	48 LH 1	30	45	GU 3	GU 6	35	..	AH

* GU indicates the left or right hypertropia was greatest up.
† Convergence near point.
‡ AH indicates alternating hyperphoria or hypertropia; AHo, alternating hypophoria or hypotropia; WR, wheel rotation; NT, not taken; R, right eye horizontal; L, left eye horizontal.

The diagnostic findings in 15 representative cases from each group have been tabulated to show the characteristics of each group.

GROUP I: *Esotropia with Bilateral Elevation in Adduction.*—These patients showed elevation of the adducted eye on lateral movements to the right or to the left. On versions in the vertical directions, these patients characteristically showed much more esotropia when looking down than up. There was also more esotropia for near than for distance. The data on 15 representative patients of this group are given in Table 1. All these patients showed more esotropia for near than for distance in the screen test. They all had more esotropia on looking down than on looking up, and they all screened a left hypertropia which was greatest up and to the right, and a right hypertropia, which was greatest up and to the left. The conver-

gence near point varied from 20 to 45 mm. Of the 11 patients in whom fusion was tested, 2 showed no fusion; 3, first-grade fusion; 5, second-grade fusion, and 1, third-grade fusion. Of the 11 patients examined with the delayed cover test, 7 showed an alternating hypertropia; 2, an alternating hypotropia; 1, a left hypotropia with the right eye horizontal; 1, a right hypertropia with the left eye horizontal, and 1, wheel rotation.

Three typical cases of this group are presented.

CASE 1 (Fig. 1).—T. S., aged 6 yr., came to the Infirmary in 1949, with a history of crossing of both eyes since the age of 3 yr.

With atropine cycloplegia, refraction was as follows:

O.D.: +2.50 sph. ⌒ +1.50 cyl., ax 100 = 20/30
O.S.: +2.50 sph. ⌣ +1.00 cyl., ax 90 = 20/30

Examination showed alternating esotropia of 20 degrees on looking straight ahead.

Vertical versions showed 5 degrees of esotropia on looking up and 40 degrees of esotropia on looking down.

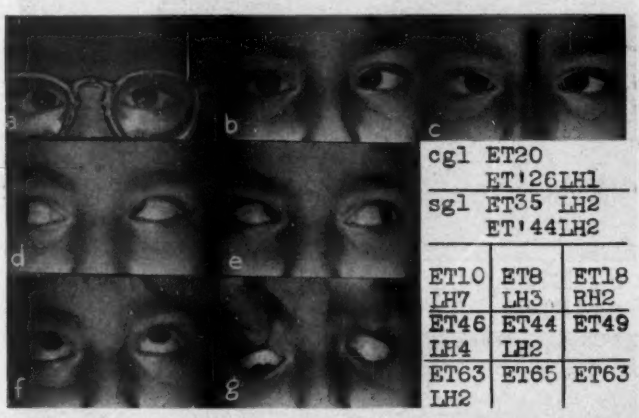


Fig. 1.—(*a*) 20 degrees of right esotropia with glasses; (*b*) 20 degrees of left esotropia without glasses; (*c*) near point of convergence, 30 mm.; (*d*) elevation of left eye in adduction, right eye fixing; (*e*) elevation of right eye in adduction, left eye fixing; (*f*) 5 degrees of esotropia, looking up; (*g*) 40 degrees of left esotropia, looking down.

Lateral versions showed bilateral elevation in adduction.

The convergence near point was 30 mm.

The delayed cover test showed alternating hypotropia.

Screening showed 20△ of esotropia for distance and 28△ of estropia for near, with 1△ of hypertropia, with glasses, and 35△ of estropia for near and 44△ esotropia for near, with left hypertropia of 2△ for distance and near without glasses.

On the synoptophore, there was first-grade fusion, with oscillation. Retinal correspondence was abnormal with the after-image test.

This case shows all the diagnostic findings of this group—more esotropia for near than for distance, more esotropia on looking down than up; the left hypertropia is to the right and greatest on looking up, and the right hypertropia is to the left and greatest on looking up. The child has a good convergence near point. One should note the small vertical measurements with the marked elevation in adduction. The elevation in adduction takes place in the extremely adducted eye, and this

position of adduction cannot be screened because the nose gets in the way and the patient cannot see the light with the adducted eye. In the position of adduction in which screening can be done, the vertical deviation is of small amount.

CASE 2 (Fig. 2.)—P. D., aged 5 yr., came to the Infirmary on Aug. 28, 1946, with a history of a crossed right eye since the age of 4½ yr. The right esotropia was intermittent when the condition first began but had become constant.

Fig. 2.—*A*: (*a*) 15 degrees of left esotropia with glasses; (*b*) 20 degrees of left esotropia and small left hypertropia without glasses; (*c*) 20 degrees right esotropia with small right hypertropia without glasses; (*d*) elevation of left eye in adduction, right eye fixing; (*e*) elevation of right eye in adduction, left eye fixing; (*f*) near point of convergence 30 mm; (*g*) 15 degrees of right esotropia looking up; (*h*) 35 degrees of right esotropia looking down.

B: (*a*) Straight eyes for distance without glasses; no hypertropia; (*b*) straight for near without glasses; no hypertropia; stereopsis; (*c*) convergence near point, 40 mm.; (*d*) eyes on same level on gaze to the right; (*e*) eyes level on gaze to left; slight limitation of right eye in adduction postoperatively; (*f*) straight on looking up; (*g*) straight on looking down.

With atropine cycloplegia, refraction was as follows:

O.D.: +3.25 sph. ⊃ +0.50 cyl., ax 90 = 20/50
O.S.: +3.00 sph. = 20/30

With occlusion, with shellac over the left lens, vision was improved to 20/30.

Orthoptic training was given for 16 treatments, without improvement.

Findings on examination (May 5, 1947) are shown in Figure 2*A*.

On the synoptophore there was second-grade fusion with amplitude; the objective and subjective angle were the same at 20Δ base out; there was right hypertropia of 2Δ. There was normal correspondence with the after-image test.

On May 5, 1947, 4 mm. recession of the right medial rectus and 7 mm. resection of the right lateral rectus muscle were performed.

The findings on postoperative examination (Oct. 26, 1950) are shown in Figure 2*B*.

This case shows all the diagnostic findings typical of Group I before operation. The right hypertropia was greater than the left hypertropia with screening. This was due to the right eye being the more dissociated eye, as it was the nondominant eye and amblyopic for a while. This finding of the greater vertical deviation in the

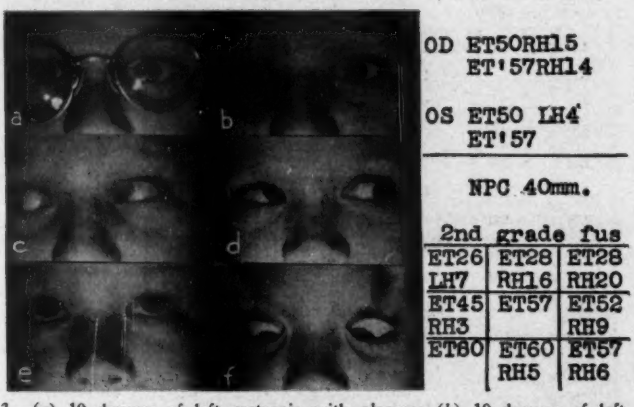


Fig. 3.—(*a*) 10 degrees of left esotropia with glasses; (*b*) 10 degrees of left esotropia without glasses, small left hypertropia; (*c*) elevation of left eye in adduction, right eye fixing; (*d*) elevation of right eye in adduction, left eye fixing; (*e*) straight on looking up; (*f*) 25 degrees of left esotropia looking down.

nondominant eye is common in cases of dissociated hypertropia. Measurements showed that the right hypertropia of 4 Δ down and to the left increased to a right hypertropia of 10 Δ up and to the left. The vertical measurements only suggest paresis of the left superior rectus with overaction of the right inferior oblique. That this was a secondary hypertropia is clear when we note that since operation on the lateral muscles only, which cured the esotropia, the elevation in adduction has completely disappeared. The vertical deviations on screening have all been eliminated except for a small amount of right hyperphoria up and to the left.

Case 3 (Fig. 3)—R. V. Z., aged 5½ yr., came to the Infirmary on Jan. 29, 1949, with a history of the eyes being crossed since the age of 18 mo.

With atropine cycloplegia, refraction was as follows:

O.D.: +3.00 sph. = 20/30
O.S.: +4.00 sph. ⊃ +1.00 cyl., ax 110 = 20/30

The findings on examination are shown in Figure 3.

This case has all the findings necessary to place it in Group I. The elevation in adduction is about the same for each eye; yet this patient screened a right hypertropia of 20 Δ and a left hypertropia of only 7 Δ. This is because, as in many cases in this group, the greatest elevation takes place in extreme adduction, a position which cannot be screened.

GROUP II: *Esotropia with Bilateral Depression in Adduction.*—The patients in this group showed depression of the adducted eye (relative elevation of the abducted eye) on lateral movements to the right or left. Clinically, this vertical deviation existed in all degrees, sometimes to such a small degree that the elevation could not be seen on lateral versions but could be measured by the prism-cover test. Table 2 shows the typical findings in 15 representative cases of Group II.

TABLE 2.—*Esotropia (in Degrees) with Bilateral Depression in Adduction*

	Esotropia Far	Esotropia Near	Esotropia Up	Esotropia Down	RH on Gaze to Right*	LH on Gaze to Left*	NPC, Mm.	Grade Fusion	Delayed Cover Test†
E. G.	34 LH 2	30 LH 4	52 LH 5	15	GD 8	GU 9	40	2d	AHo
C. H.	60 RH 1	48	47 LH 2	37	GU 8	GU 10	40	2d	AHo
A. F.	36	26	50 RH 2	43	GU 4	GU 3	50	2d	AHo
A. S.	23 RH 6	23 RH 4	39	0 LH 2	GD 20	GD 12	35	3d	AHo
D. K.	20 RH 20	16 RH 8	26	0 LH 9	GD 7	GD 10	40	2d	AH WR
L. E.	52 LH 1	52 LH 5	48 LH 6	40	GU 7	GU 5	70	No	AHo
M. J.	37 LH 4	20 LH 2	50	10 LH 4	GD 13	GD 16	40	3d	AH WR
N. P.	63 LH 6	59	65	45 LH 4	GD 6	GD 6	40	..	AHo
A. D.	23 LH 4	19 LH 4	28	12 RH 1	GD 17	GD 4	90	..	NT
B. H.	60 RH 6	30 LH 4	36 LH 11	25 LH 7	GU 4	GD 18	65	..	NT
E. L.	38 LH 8	30 LH 8	22	16	GU 12	GD 24	30	2d	Ind.
C. H.	31	31 RH 2	40	24 LH 2	GU 2	GU 2	35	3d	NT
O. L.	70	67	62	56	GU 4	GU 4	40	..	AH
R. N.	55 LH 2	51	49 LH 4	48	GU 2	GU 2	45	..	NT
R. S.	38 LH 8	37 LH 10	47 LH 10	12 RH 7	GD 13	GD 10	40	..	NT

* GU indicates hypertropia greatest up, and GD, hypertropia greatest down.
† As in table 1; Ind. means "indefinite."

Table 2 shows that 11 patients screened more esotropia for distance than for near, 4 screened the same amount of esotropia for distance and for near, and none screened less for distance than for near. All the patients screened more estropia on looking up than on looking down. They all had elevation of the right eye on gaze to the right and elevation of the left eye on gaze to the left. Six patients had the greatest vertical deviation up; six, the greatest vertical deviation down, and three, the greatest vertical deviation up on the right and down on the left. The near point of convergence varied from 30 to 90 mm. Of the nine for whom fusion was tested, one had no fusion, five had second-grade fusion, and three had third-grade fusion. Of the 10 patients on whom the delayed cover test was done, 6 showed an alternating hypotropia; 3 an alternating hypertropia, 2 of whom also had wheel rotation, and for 1 the findings were indefinite.

Three illustrative cases of this group are presented.

CASE 4 (Fig. 4).—M. J., aged 20, came to the Infirmary in 1949, with a history of the eyes having been crossed at times since birth.

With atropine cycloplegia, refraction was as follows:

O.D: +3.00 sph. = 20/20
O.S.: +3.00 sph. = 20/20

Fig. 4.—(*a*) Eyes straight for near with glasses; (*b*) eyes straight for near without glasses; (*c*) 20 degrees of left esotropia for distance; (*d*) depression of left eye in adduction, right eye fixing; relative elevation of right eye; (*e*) depression of right eye in adduction; relative elevation of left eye; (*f*) 20 degrees of esotropia looking up; (*g*) 5 degrees of right exotropia looking down.

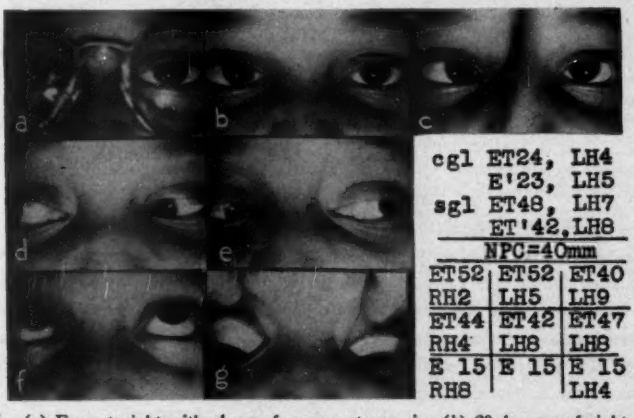


Fig. 5.—(*a*) Eyes straight with glasses for near, stereopsis; (*b*) 20 degrees of right esotropia for distance without glasses; (*c*) near point of convergence, 20 mm.; (*d*) depression of left eye in adduction; (*e*) depression of right eye in adduction; (*f*) 20 degrees of right esotropia looking up; (*g*) 10 degrees of exotropia looking down.

Examination showed 20 degrees of esotropia for distance and for near at times; the eyes could be straight for distance and near (Fig. 4).

The delayed cover test showed alternating hyperphoria and wheel rotation in.

Fusion was third grade at zero.

This case shows all the findings typical of Group II, viz., an esotropia for distance and only an esophoria for near, a marked esotropia on looking up, and a slight divergence on looking down. The left hyperphoria is to the left and greatest in looking down, and the right hyperphoria is to the right and greatest in looking down. She screened an esophoria of 10 D. on looking down, but the photographs show a definite exotropia. This discrepancy is explained by the impossibility of screening the eyes when rotated down so far.

CASE 5 (Fig. 5).—E. G., aged 11 yr., came to the Infirmary in 1948, with a history of crossed eyes at birth.

With atropine cycloplegia, refraction was as follows:

O.D.: +3.50 sph. ⌒ +3.25 cyl., ax 85 = 20/50
O.S.: +3.50 sph. ⌣ +1.75 cyl., ax 90 = 20/50

The findings on examination are shown in Figure 5.

The delayed cover test showed alternating hypophoria.

Fusion was third grade at the objective angle.

This case has all the typical findings of Group II. The patient has bilateral depression in adduction with relative elevation of each eye in abduction. In her screening measurements she has the left hyperphoria to the left and the right hyperphoria to the right. However, the greatest right hyperphoria is down and to the right, and the greatest left hyperphoria is up and to the left.

CASE 6 (Fig. 6*A*).—C. H., aged 12, came to the Infirmary in 1949, with a history of crossed eyes since birth.

With atropine cycloplegia, refraction was as follows:

O.D: +1.25 sph. ⌒ +1.25 cyl., ax 65 = 20/50
O.S.: +0.25 sph. ⌣ +1.00 cyl., ax 105 = 20/30

Examination showed 20 degrees of esotropia with and without glasses.

Vertical versions showed 20 degrees of esotropia on looking up and straight eyes on looking down.

Lateral versions showed marked bilateral depression in adduction.

The delayed cover test showed alternating hypotropia and fine horizontal nystagmus.

Fusion was third grade at the objective angle; there was normal correspondence with the after-image test.

At operation, on May 31, 1950, recession of 5.5 mm. was performed on both medial rectus muscles.

The findings on postoperative examination (Aug. 2, 1950) are shown in Figure 6*B*.

The delayed cover test showed alternating hypertropia.

Before operation, this patient had all the findings which placed her in Group II. With the large amount of depression that she showed, larger vertical screening measurements would be expected. Actually, in screening she showed a small bilateral hypertropia in the characteristic directions for this group. Considering the marked depression in adduction, along with the increase in vertical measurements up, the diagnosis could be bilateral paresis of the inferior oblique with overaction of the superior oblique muscles.

After a bilateral recession of the medial rectus, of 5.5 mm., her eyes became divergent. Now she can be classified under Group IV—exotropia with bilateral depression in adduction. She has more exotropia on looking down, with straight eyes to a slight divergence on looking up. The characteristic left hypertropia is to the left and greatest down, and the right hypertropia is to the right and greatest down. There has been a considerable increase in the vertical measurements since

operation. The left hypertropia has increased from 3 to 30 Δ on looking down and to the left, and the right hypertropia has increased from 0 to 34 Δ on looking down and to the right. If we had seen this girl now for the first time, and did not take into consideration her history and other findings characteristic of the group, we should make a diagnosis of bilateral paresis of the inferior rectus muscles with overacting superior obliques, on the basis of the screening measurements alone. The pronounced vertical deviations that developed after the operation on the horizontal

Fig. 6.—*A:* (*a*) 20 degrees of right esotropia for near; (*b*) 20 degrees of left esotropia for near; (*c*) near point of convergence, 25 mm.; (*d*) depression of left eye in adduction, right eye fixing; (*e*) depression of right eye in adduction, left eye fixing; (*f*) 25 degrees right esotropia, looking up; (*g*) eyes straight, looking down.

B: (*a*) 5 degrees of right exotropia for distance; (*b*) 15 degrees of left exotropia for near; (*c*) near point of convergence, not elicited; (*d*) eyes straight looking up; (*e*) 30 degrees of right exotropia looking down; (*f*) depression of left eye in adduction, right eye fixing; (*g*) depression of right eye in adduction, left eye fixing.

muscles only, and the change in position of the screening measurements of the hypertropia, which have become greatest down, whereas before operation they were greatest up, are evidence that the vertical deviation is secondary to the exotropia.

GROUP III: *Exotropia with Bilateral Elevation in Adduction.*—These patients showed elevation of the adducted eye on lateral movements to the right or to the left. Clinically, this elevation existed in all degrees, and in some patients to such a small degree that it could not be seen on lateral versions but could be measured by the prism-cover test. Table 3 shows the typical findings for 15 patients in this group. This table shows that the screening measurements for eight patients were greater for distance than for near. All 15 patients had an exotropia for distance but an exophoria for near, and in seven patients the exophoria for near measured more than the exotropia for distance. Every patient showed an exotropia on looking up and a smaller exophoria on looking down. Every patient screened a left hyperphoria on gaze to the right, greatest up, and a right hyperphoria on gaze to the

TABLE 3.—*Exotropia (in Degrees) with Bilateral Elevation in Adduction*

	Exotropia Far	Exotropia Near	Exotropia Up	Exotropia Down	RH on Gaze to Right	LH on Gaze to Left	NPC, Mm.	Grade Fusion	Delayed Cover Test
G. Z.	18 LH 11	4	48	3	GU 9	GU 15	40	3d	NT
J. L.	24	16	37	28	GU 7	GU 7	20	3d	NT
N. P.	14 RH 2	20 RH 1	28	24	GU 12	GU 7	30	3d	AH
J. W.	25 LH 7	7	12	3	GU 7	GU 9	20	3d	AH
P. D.	3	2	5 RH 4	0	GU 8	GU 20	25	3d	N*
N. M.	10	16	18	10	GU 2	GU 2	35	3d	NT
P. G.	34 RH 12	38 RH 12	48 RH 6	28	GU 14	GU 14	50	3d	AH
F. P.	16 LH 8	20 LH 5	50 LH 10	4	GU 28	GU 20	20	2d	N
P. W.	32 RH 4	37	48 LH 4	9	GU 10	GU 14	30	2d	AHo
S. U.	34 LH 4	42 LH 5	46 LH 4	40	GU 12	GU 4	60	..	NT
H. F.	9 LH 2	34 LH 1	30	20	GU 2	GU 8	35	2d	NT
R. N.	27	20	25	8	GU 4	GU 5	55	2d	NT
L. S.	23 LH 5	12 LH 1	33 LH 4	10 LH 4	GU 18	GU 7	50	2d	NT
J. C.	9	4	7 RH 2	4 RH 2	GU 1	GU 3	20	3d	N
L. P.	58 LH 8	44 LH 2	68 LH 16	45	GU 25	GU 10	35	..	NT

*N indicates no deviation.

left, greatest up. The convergence near point was good in every case, varying from 20 to 50 mm. Every patient for whom fusion was tested (13) had second or third-grade fusion. The delayed cover test was done on seven patients in this group, and in three no deviation was found; in three there was an alternating hyperphoria, and in one, an alternating hypophoria.

Three illustrative cases of this group are presented.

CASE 7 (Fig. 7).—M. S., aged 9 yr., came to the Infirmary in 1949, with a history that she could not see the blackboard at school and that her eyes turned out at times.

With atropine cycloplegia, refraction was as follows:

O.D.: —0.50 sph. $\bigcirc$ +1.00 cyl., ax 95 = 20/30
O.S.: —1.50 sph. $\bigcirc$ +1.75 cyl., ax 75 = 20/30

Screening measurements showed exotropia of 34△, with right hypertropia of 12△, for distance, and exophoria of 48△, with right hypertropia of 16△, for near.

Fusion was third grade.

This case has all the findings characteristic of Group III. When the patient's eyes are dissociated with the cover test, each eye moves out and up under cover. The vertical deviation then is due to the horizontal dissociation of the eyes, since her eyes are straight when not dissociated. Examination of the vertical screening measurements shows that on gaze to the right she has opposite vertical measurements: 4 Δ of right hypertropia down, and 3 Δ of right hypertropia laterally, but 14 Δ of left hypertropia characteristically up and to the right. On gaze to the left the 8 Δ of right hyperphoria down and to the left increases to 24 Δ of right hypertropia straight to left and then decreases to 15 Δ of right hypertropia up and to the left. These variations in the screening measurements speak for secondary vertical deviations, and not symmetrical paretic vertical muscles. Paretic vertical muscles have one type of vertical deviation in lateral gaze, which increases or decreases symmetrically in upward or downward gaze.

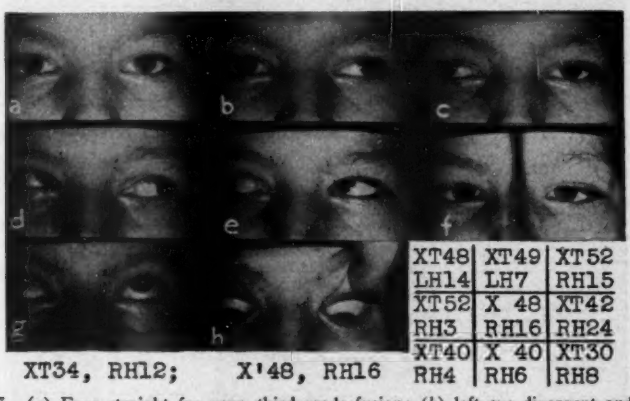


Fig. 7.—(*a*) Eyes straight for near, third-grade fusion; (*b*) left eye divergent and elevated under cover, right eye fixing; (*c*) right eye divergent and elevated under cover, left eye fixing; (*d*) elevation of right eye in adduction, left eye fixing; (*e*) elevation of left eye in adduction, right eye fixing; (*f*) near point of convergence, 30 mm.; (*g*) 15 degrees right exotropia on upward gaze; (*h*) 5 degrees exotropia looking down.

Case 8 (Fig. 8*A*).—M. B., aged 20, came to the Infirmary in 1949, with a history that both eyes had turned out since birth.

With atropine cycloplegia, refraction was as follows:

O.D: —1.00 sph. = 20/20
O.S.: —1.00 sph. = 20/20

Examination showed 25 degrees of alternating exotropia for distance and near.

Vertical versions showed marked exotropia on looking up and down, being slightly greater up.

Lateral versions showed a slight suggestion of elevation in adduction.

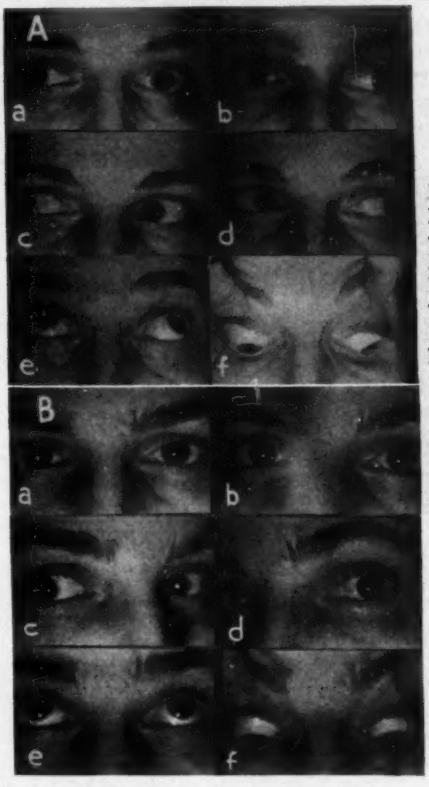
The convergence near point could not be obtained.

Fusion was first grade, with oscillation. The objective angle was 48 D. base in, with a left hypertropia of 3Δ; it was impossible to get third-grade fusion.

At operation (Aug. 24, 1949) 8 mm. of recession was done on both lateral rectus muscles and 8 mm. of resection on the left medial rectus muscle.

The findings on postoperative examination (June 16, 1950) are shown in Figure 8*B*.

This case has enough findings to place it in Group III. Before operation the patient had a left hypertropia of 24 Δ up and to the right in the diagnostic direction and position of the group. She also had a right hypertropia of 4 Δ down and to the right. This combination of opposite vertical deviations is found frequently in patients with secondary vertical deviations. After operation on the lateral muscles only, her eyes became straight in all positions with a phoria on screening only. Stereopsis developed after operation, with a near point of convergence of 150 mm.



cgl XT52,LH5 XT'66,LH5		
sgl XT46,LH4 XT'62,LH5		
XT70 LH24	XT60 LH14	XT63 RH5
XT44 LH9	XT62 LH4	XT56
XT52 RH4	XT52	XT56 LH4

X 18 LH4	X 14	X 18 RH4
X 6	X 8 RH1	X 8 LH4
X 8	X 8	X 6
cgl ortho X'8RH1		
NPC 150mm 3rd grade fus		

Fig. 8.—*A:* (*a*) 20 degrees of right exotropia for near; (*b*) 25 degrees of left exotropia for near; (*c*) slight elevation of left eye in adduction with limitation of left eye in adduction, right eye fixing; (*d*) eyes level in lateral gaze with limitation of right eye in adduction; (*e*) 30 degrees of left exotropia, looking up; (*f*) 25 degrees of left exotropia, looking down.

B: (*a*) Eyes straight for near, third-grade fusion; (*b*) convergence near point, 150 mm.; (*c*) eyes level on gaze to right, some limitation of right eye in abduction; (*d*) eyes level on gaze to left, some limitation of the left eye in abduction; (*e*) eyes straight looking up; (*f*) eyes straight looking down.

The marked reduction of the left hypertropia of 24 Δ to one of 3 Δ with recession of the lateral muscles only is evidence that the vertical deviations were secondary to the exotropia.

CASE 9 (Fig. 9).—P. G., aged 6 yr., came to the Infirmary in March, 1950, with a history that the eyes had turned out intermittently since birth.

With atropine cycloplegia, refraction was as follows:

O.D.: —1.00 sph. ⌒ +1.00 cyl., ax 90 = 20/30
O.S.: —0.50 sph. ⌒ 0.50 cyl., ax 85 = 20/30

The findings on examination are shown in Figure 9*A*.

Delayed cover test showed alternating hyperphoria.

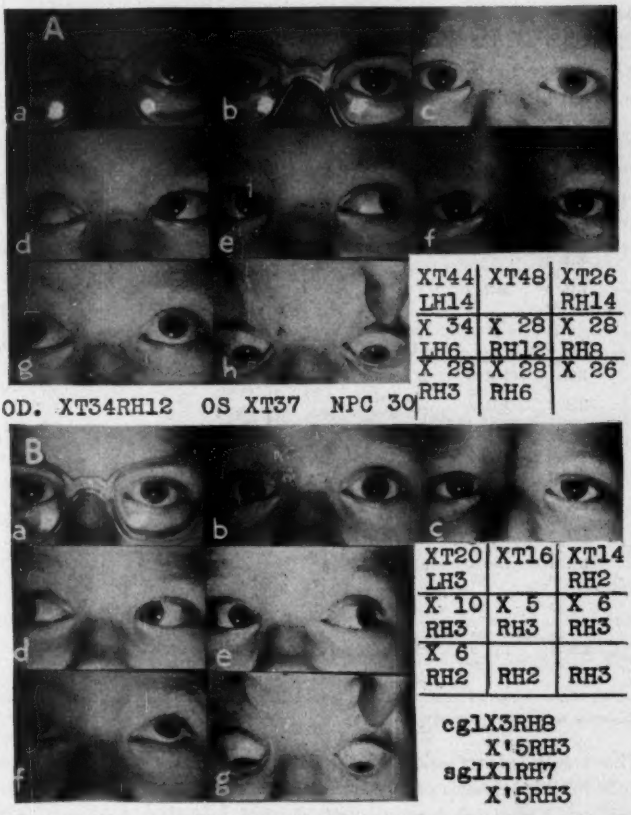


Fig. 9.—*A:* (*a*) 20 degrees of right exotropia for distance with glasses; (*b*) eyes straight for near with glasses; third-grade fusion; (*c*) eyes straight for near without glasses; (*d*) elevation of left eye in adduction, right eye fixing; (*e*) elevation of right eye in adduction, left eye fixing; (*f*) convergence near point, 50 mm.; (*g*) 35 degrees of right exotropia looking up; (*h*) eyes straight looking down.

B: (*a*) Eyes straight for distance with glasses; (*b*) eyes straight for near without glasses; (*c*) convergence near point, 50 mm.; (*d*) eyes level on gaze to right; (*e*) eyes level on gaze to left; (*f*) 5 degrees of exotropia looking up; (*g*) eyes straight looking down. Note that the elevation in adduction cannot be seen postoperatively, yet can be found with prism-cover measurements.

At operation (Aug. 3, 1950) 7 mm. recession was performed on both lateral rectus muscles.

The findings on postoperative examination (Sept. 21) are shown in Figure 9*B*.

Delayed cover test showed right hyperphoria only.

On examination (Nov. 29, 1950) the eyes were straight in all positions.

Delayed cover test showed orthophoria, no hyperphoria.

Fusion was third grade.

This case has all the findings diagnostic of Group III. Examination of the vertical screening measurements only would speak for bilateral paresis of the superior rectus with overaction of the inferior oblique muscles. The finding that differentiate the secondary vertical deviation which she had from paresis of the vertical muscles are the following:

1. On gaze to the right she had a right hyperphoria of 3 Δ down and to the right, with left hyperphoria of 14 Δ up and to the right.

TABLE 4.—*Exotropia (in Degrees) with Bilateral Depression in Adduction*

	Exotropia Far	Exotropia Near	Exotropia Up	Exotropia Down	RH on Gaze to Right	LH on Gaze to Left	NPC, Mm.	Grade Fusion	Delayed Cover Test
H. E.	9 RH 2	30	12	22	GD 4	GD 2	180	3d	NT
R. F.	30 RH 6	54 RH 9	21	44	GD 18	GD 12	35	2d	AH
B. J.	50	56 LH 8	50	66	GD 9	GD 20	No	No	NT
H. T.	40 RH 4	56	42 LH 2	50 LH 3	GD 4	GD 7	No	2d	NT
C. C.	32 RH	42 RH 5	42 LH 3	50 RH 9	GD 12	GD 6	No	No	NT
D. B.	9 RH 6	25 RH 6	10 RH 4	45	GD 12	GD 30	120	No	AH WR ON*
R. G.	30 LH 5	22 LH 5	10 LH 2	46 RH 3	GD 12	GD 20	No	No	NT
J. S.	10 RH 14	22 RH 14	12 RH 6	20 RH 12	GD 22	GD 20	No	..	AH
D. N.	32 RH 6	47 RH 4	44	46	GD 4	GD 4	120	2d	AH
B. N.	47 LH 6	56 LH 4	43	56	GD 5	GD 7	No	..	AH
R. G.	37 LH 4	44 LH 2	48 LH 2	49	GD 7	GD 12	150	2d	AH
C. P.	40	50	40	50	GD 8	GD 8	No	..	AH ON
A. D.	19	36	18	41 RH 4	GD 4	GD 4	90	3d	Ind.
C. K.	22 LH 10	40 LH 9	30 LH 6	45 RH 10	GD 12	GD 35	90	..	AH
L. C.	64	66	48 LH 2	100 LH 20	GD 24	GD 45	90	..	AH

* ON indicates occlusion nystagmus.

2. The results of recession of the lateral muscles correcting the exotropia reduced the left hyperphoria of 14 Δ to one of 3 Δ and the right hypertropia of 14 Δ to one of 2 Δ. This is evidence that the vertical deviations were secondary to the exotropia.

3. The disappearance of the alternating hyperphoria in the delayed cover test after the cure of the exotropia is evidence that the alternating vertical deviations were secondary to the dissociation of the eyes by the exotropia.

GROUP IV: *Exotropia with Bilateral Depression in Adduction.*—These patients show depression of the adducted eye (relative elevation of the abducted eye) on lateral movements. Table 4 shows the typical findings for 15 representative patients in this group. From Table 4 it can be seen that every patient screened a

larger degree of exotropia for near than for distance. The exotropia on looking down was greater than the exotropia on looking up in every case. Each patient screened a right hypertropia on gaze to the right and a left hypertropia on gaze to the left, which was greatest down. The near point of convergence varied from zero, for seven patients, to poor for seven others with one patient having a convergence near point of 35 mm. Of the 10 patients whose fusion was tested, four had no fusion, four had second-grade fusion, and two had third-grade fusion. Of the nine patients on whom the delayed cover test was done, the findings in one were indefinite; in the remaining eight there was an alternating hypertropia. One of the latter eight patients showed wheel rotation along with the alternating hypertropia, and one

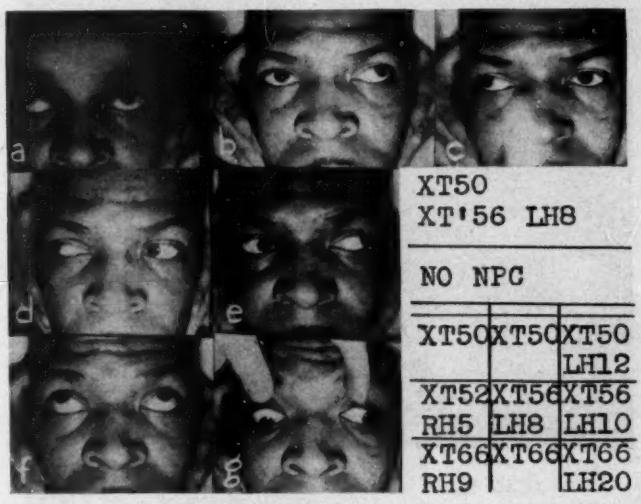


Fig. 10.—(*a*) 45 degrees of right exotropia for distance; (*b*) 45 degrees of left exotropia for near; (*c*) no convergence near point; (*d*) depression of left eye in adduction, right eye fixing; (*e*) depression of right eye in adduction, left eye fixing; (*f*) 25 degrees of right exotropia on upward gaze; (*g*) 65 degrees left exotropia looking down.

showed both wheel rotation and occlusion nystagmus with the alternating hypertropia.

Case 10 (Fig. 10).—B. J., aged 31, came to the Infirmary in 1949, with a history of crossed eyes since birth.

With atropine cycloplegia, refraction was as follows:

O.D.: +0.50 sph. ⊃ +0.25 cyl., ax 90 = 20/70
O.S.: +0.37 sph. ⊃ +0.25 cyl., ax 65 = 20/30

Examination (Fig. 10) showed all the characteristics of the group.

Case 11 (Fig. 11).—R. F., aged 31, came to the Infirmary in 1949, with a history of the eyes having turned out at times.

With homatropine cycloplegia, refraction was as follows:

O.D.: —0.75 sph. = 20/20
O.S.: plano = 20/20

The findings on examination are shown in Figure 11.

Delayed cover test showed alternating hyperphoria.

This case has all the characteristic findings of Group IV. The patient has mostly an exophoria, which becomes an intermittent exotropia. When his eyes are straight, he has stereopsis at zero, showing that such large screening measurements can indicate a phoria.

CASE 12 (Fig. 12*A*).—R. G., aged 5 yr., came to the Infirmary in 1945, with the history that his right eye had turned out and up at times since birth, especially when he was tired or daydreaming.

With atropine cycloplegia, refraction was as follows:

O.D.: +2.25 sph. = 20/30
O.S.: plano = 20/20

Examination showed that the eyes could be straight for distance and near; when they were dissociated with a cover, he had 20 degrees of alternating exotropia.

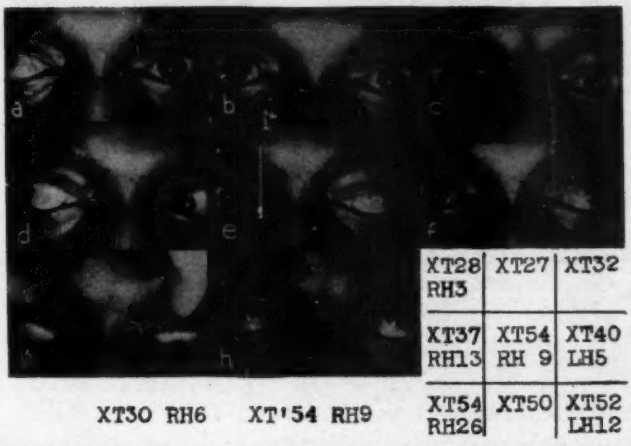


Fig. 11.—(*a*) 30 degrees of right exotropia, 10 degrees of right hypertropia; (*b*) eyes straight for near; (*c*) convergence near point, 30 mm.; (*d*) depression of left eye in adduction, right eye fixing; (*e*) depression of right eye in adduction, left eye fixing; (*f*) 25 degrees of left exotropia, 5 degrees of left hypertropia under cover, right eye fixing; (*g*) 30 degrees of right exotropia looking down; (*h*) 5 degrees of left exotropia looking up.

Vertical versions showed straight eyes on looking up and 25 degrees of exotropia on looking down.

Lateral versions showed bilateral depression in adduction.

The convergence near point was 12 inches (30 cm.).

Delayed cover test showed alternating hyperphoria.

Fusion was third grade at the subjective and objective angles, which were 36△ of exotropia, with 4△ of left hypertropia.

At operation (Sept. 14, 1949) 8 mm. recession was performed on both lateral rectus muscles.

The findings on postoperative examination (Sept. 6, 1950) are shown in Figure 12*B*.

This case had all the findings for inclusion in Group IV before operation. The photographs show the exotropia and hypertropia when the eyes were dissociated with a cover. When his eyes were straight, no vertical deviation was seen. Examination of the versions showed that the eyes were depressed in adduction. A diagnosis

of bilateral paralysis of the inferior rectus with overacting superior obliques might be made if only the versions and screening measurements were taken into consideration. The differential factors placing this case in the group of dissociated vertical deviations are as follows:

1. Straight eyes with second-grade fusion. The vertical deviation can be measured and seen only when the eyes are dissociated into an exotropia with the cover test.

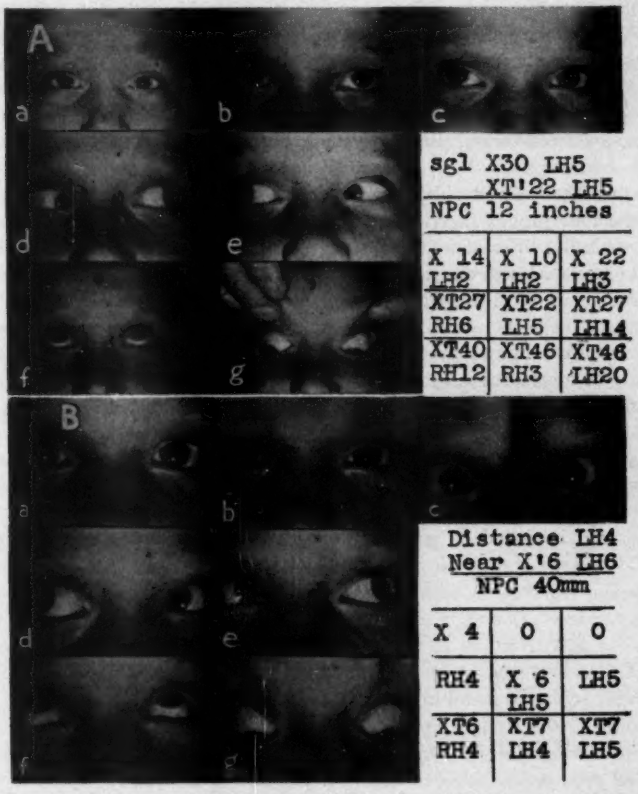


Fig. 12.—*A:* (*a*) Eyes straight for near; (*b*) elevation and divergence of left eye under cover; (*c*) elevation and divergence of right eye under cover; (*d*) depression of right eye in adduction, left eye fixing; (*e*) depression of left eye in adduction, right eye fixing; (*f*) eyes straight, looking up; (*g*) 30 degrees of right exotropia, looking down.

B: (*a*) Eyes straight for near, stereopsis; (*b*) eyes straight to 5 degrees esotropia for distance; (*c*) convergence near point, 40 mm.; (*d*) eyes level on gaze to the right; (*e*) eyes level on gaze to left; (*f*) eyes straight, looking up; (*g*) 10 degrees of left exotropia, looking down.

Note that the depression in adduction cannot be seen after operation. The hyperphoria is brought out with prism-cover measurements.

2. The combination of the opposite vertical deviations on gaze to the right—2 Δ of left hypertropia up and to the right, and 12 Δ of right hypertropia down and to the right.

3. Improvement in the left hypertropia from 20 Δ to 5 Δ and in the right hypertropia from 12 Δ to 4 Δ, and improvement in the depression in adduction by correction of the intermittent exotropia after operation on the lateral rectus muscles are evidence that the vertical deviations were secondary to the dissociation of the eyes by the exotropia.

COMMENT

Bilateral elevation in adduction in cases of esotropia, Group I, has been discussed in the literature by many workers. In cases in which this evaluation has been prominent, White [12] considered the cause to be a bilateral overaction of the inferior obliques due to bilateral paresis of the superior rectus muscles. Chavasse [1] called this a secondary cyclovertical deviation; Guibor [13] referred to it as esotropia with synkinetic overaction of the inferior obliques, and Cords [14] called it strabismus convergens sursoadductorius. Bielschowsky [15] explained it as due to overaction of the inferior obliques; when the eyes are dissociated, the inferior oblique, being a stronger muscle than the superior oblique, elevates the eye in adduction.

In the four groups described, the elevation or depression in adduction can be found, clinically, to exist in various degrees. In some cases the vertical deviation may be present in such a small degree that it cannot be recognized on lateral versions, but it can be measured by the prism-screen test (Figs. 8*A*, *B* and 9*B*). In other cases the elevations can be pronounced on lateral versions, and yet the vertical deviations measured may be very small. These small vertical measurements would ordinarily be considered insignificant in the presence of such large lateral deviations, but in these groups the direction, right or left, and position, up or down, not the amount of the vertical deviation, is of diagnostic value. A right or left hypertropia or hyperphoria in the proper direction and position, regardless of its amount, helps place the case in the proper group (Figs. 6, 8*B*, *1* and *2*). Some cases have all the other features which would place them in the proper group; yet only one vertical deviation can be found on screening. The vertical deviation becomes significant when it is greatest in the typical direction and position for the diagnosis of the group.

In young children on whom screening measurements cannot be made, the versional movements are used to classify the squint. In certain cases the lateral versions do not show much, but the vertical versions are rather consistent in that the greater squint is found on looking up in Groups II and III, whereas in Groups I and IV the greatest squint is found on looking down.

A study of the straight-up and straight-down position also gives one an idea of the binocular status of the eyes. If the eyes are straight in one of these positions, fusion and good vision can be maintained. The finding of a high incidence of second- and third-grade fusion with a good convergence near point in cases in Groups II and III (Tables 2 and 3) bears out this statement. If the eyes are straight at times in these positions, fusional amplitudes could be developed. Orthoptic training could

12. White, J. W.: Hyperphoria Diagnosis and Treatment, Arch. Ophth. **8**:739 (May) 1932.

13. Guibor, G. P.: Synkinetic Overaction of the Inferior Oblique Muscle, Am. J. Ophth. **32**:221, 1949.

14. Cords, R., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Opthalmologié, Heidelberg, Springer-Verlag, 1922, Vol. 13, p. 158.

15. Bielschowsky, A.: Über die relative Ruhelage der Augen, Ber. Versamml. deutsch. ophth. Gesellsch. **13**:67, 1913.

be started in the position in which the eyes are straight, and with the development of good fusion amplitude the squint could become intermittent. In such cases the prognosis for a functional cure would be excellent.

Evidence That the Vertical Deviations Are Secondary to the Lateral Squint.—If the diagnosis of paresis of the vertical muscles were to be made in these cases, the commonest muscles involved would be the superior rectus muscles, with paralysis of the inferior rectus muscles next in frequency; the superior and inferior obliques would rarely be affected.[16] The nuclei of the branches of the third cranial nerve supplying the superior and inferior rectus muscles are closely grouped with the other nuclei of the third nerve, in the floor of the aqueduct of Sylvius. It is extremely unlikely that these isolated bilateral involvements could occur so frequently. It is also improbable that these isolated involvements could occur without any other implication of the third-nerve complex. Developmentally and anatomically, the levator muscles are closely related to the superior rectus muscles, and the inferior obliques to the inferior rectus muscles. It is improbable that such frequent bilateral involvements of a vertical rectus muscle could be peripheral without affecting the levators or the inferior obliques at the same time. The most reasonable assumption on an anatomical basis is that the vertical deviations are secondary to the dissociation of the eyes.

Certain patients show marked elevation or depression in adduction but have small screening measurements. If these vertical deviations were due to paretic vertical rectus muscles, they would be greatest when the affected eyes were abducted 23 degrees and could be readily screened in this position. Secondary deviations are greatest in many cases in extreme adduction, that is, when the eyes are rotated laterally so that the fixing eye is in extreme abduction (Figs. 1, 3 and 6). The vertical measurements are small because the eyes cannot be screened in this position.

The origin of secondary deviations can be seen very clearly in certain cases of acute paralysis of the lateral rectus. In these cases there developed vertical components and, at times, dissociated vertical divergences. Cure of the lateral squint resulted in the disappearance of the hypertropias.[17]

The etiology of vertical deviations can also be seen in some cases of horizontal squint with amblyopia. When the good eye is occluded, a vertical component develops, which was not present before the occlusion. The etiologic factor here would be the Marlow effect on eyes with strabismus.

Patients with secondary deviations do not have torticollis and are able to fuse large vertical deviations; this would be rare in a case of primary vertical paralysis. These patients can have straight eyes with third-grade fusion, as can be seen in the photographs; yet, when the eyes are dissociated, they have horizontal deviations with large vertical components.[18] The vertical components are usually alternating hyperphorias or hypertropias (Figs. *7A*, 11 and *12A*).

16. White, J. W.: What Is the Minimum Routine Examination of Muscles? Arch. Ophth. **24**:1113 (Dec.) 1940.

17. Guibor, G.: Strabismus Ophthalmic Symposium, St. Louis, C. V. Mosby Company, 1950, p. 236.

18. Costenbader, F.: Strabismus Ophthalmic Symposium, St. Louis, C. V. Mosby, Company, 1950, p. 361.

In all our cases in these groups the vertical-component measurements were consistently smaller than those of the horizontal squint. This finding would indicate that the vertical deviations were secondary.

Many patients in these groups show the typical vertical deviation in the proper direction and position; however, an opposite vertical deviation will be found in the same direction, but in the opposite position. This combination of opposite vertical deviations on the same side cannot be due to paretic vertical muscles, since in these cases the vertical deviation is usually the same on one side and increases in upward or downward gaze (Figs. 3, 7, 8, 9 and 12).

Unpredictable and poor results of vertical-muscle surgery only in correcting vertical deviations in cases in these groups has been the rule. This has also been the experience of other workers.[19] Good results of correct lateral-muscle surgery in improving the vertical deviations is evidence that these vertical deviations were secondary (Figs. 2, 8, 9 and 12) (*A* and *B*).

Reports from the literature of the marked improvement in, or cure of, the vertical component when the lateral deviations were straightened by surgical correction, glasses, or orthoptic training suggests that the vertical deviations in these cases were secondary.[20]

SUMMARY

In a study of 615 patients with horizontal squint, 79% were found to have a vertical component. Fifty per cent of the patients with a vertical component could be classified into four clinical entities:

Group I: Esotropia with bilateral elevation in adduction. In these patients the esotropia is greater for near and downward gaze than for distance and upward gaze; a right hypertropia is present on gaze to the left, and a left hypertropia, on gaze to the right. The convergence near point is very good.

Group II: Esotropia with bilateral depression in adduction. In these patients the esotropia is greater for distance and upward gaze than for near and downward gaze; a right hypertropia is present on gaze to the right, and a left hypertropia, on gaze to the left. The convergence near point is good to fair.

Group III: Exotropia with bilateral elevation in adduction. In this group the exotropia is greater for distance and upward gaze than for near and downward gaze; a right hypertropia is present on gaze to the left, and a left hypertropia, on gaze to the right. The convergence near point is good.

Group IV: Exotropia with bilateral depression in adduction. In these patients the exotropia is greater for near and downward gaze than for distance and upward

19. Chavasse.[1] Costenbader, F., in Strabismus Ophthalmic Symposium, St. Louis, C. V. Mosby Comapny, 1950, p. 373.

20. Guibor, G. P.: Strabismus Ophthalmic Symposium, St. Louis, C. V. Mosby Company, 1950, p. 236. Bielschowsky.[18] Swan, K.: Strabismus Ophthalmic Symposium, St. Louis, C. V. Mosby Company, 1950, p. 458. Grant, H. W., in discussion on Dunnington, J. H., and Wheeler, M. C.: Operative Results in 211 Cases of Convergent Strabismus, Arch. Ophth. **38**:9 (July) 1942. Scobee, R. C., in Strabismus Ophthalmic Symposium, St. Louis, C. V. Mosby Company, 1950, p. 331. White, J. W.: Indications for Treatment for Combined Lateral and Vertical Strabismus, Arch. Ophth. **10**:585 (Nov.) 1933. Brown, H. W.: Strabismus Symposium, St. Louis, C. V. Mosby Company, 1950, p. 213.

gaze; a right hypertropia is present on gaze to the right, and a left hypertropia, on gaze to the left. The convergence near point is usually poor.

The vertical deviation in these cases are considered to be secondary to the horizontal squint, and evidence is presented to show that they can be secondary. The differentiation from vertical deviations due to paralysis of individual vertical muscles is based on anatomical factors, variations found on screening, and results of lateral-muscle surgery only.

The importance of this classification is a practical one, because in cases which fall into these groups the horizontal squint is corrected first. If the correction of the laterial squint is successful, whether by glasses, orthoptics, or operation, the vertical deviation will be much improved or will completely disappear.

Dr. Thomas Allen provided the camera for use in the Motility Clinic, and Miss Lydia Heinen made the screening measurements and did the diagnostic work on the major amblyoscope.

EFFECT OF SYSTEMIC CORTISONE AND CORTICOTROPHIN (ACTH) ON EXPERIMENTAL HERPES SIMPLEX KERATITIS

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IRVING H. LEOPOLD, M.D.
AND
CHARLES G. STEINMETZ, M.D.
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IN CONTRAST with the spectacular recovery, without relapse, reported by Fitzgerald and associates [1] in four cases of dendritic ulcer of the cornea treated by local instillations of cortisone, Scheie, Tyner, and associates [2] found questionable improvement in two cases of keratitis metaherpetica, and Steffensen, Wishbow, and associates [3] observed subjective relief, but incomplete healing, in two cases of dendritic keratitis. Leopold, Purnell, and co-workers [4] treated four patients with dendritic keratitis by daily subconjunctival injections of cortisone and obtained partial improvement in three. In experimental herpes simplex keratitis in the rabbit, Hallett, Leopold, and associates [5] found no beneficial effect from treatment with cortisone drops and ointment, and actually a deleterious effect from subconjunctival injection of cortisone.

Steffensen and colleagues [3] stated that diseases of viral etiology in general have been unresponsive to corticotrophin (ACTH) and cortisone. This observation is further borne out by the failure of Loosli and associates [6] to demonstrate any beneficial influence of corticotrophin on the course of experimental influenzal-

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From the Department of Research, Wills Hospital.

This investigation was supported by a research grant from the National Institutes of Health, United States Public Health Service.

1. Fitzgerald, J. R., and others: Early Clinical Results of ACTH and Cortisone Treatment of Ocular Diseases, A. M. A. Arch. Ophth. **45**:320 (March) 1951.

2. Scheie, H. G.; Tyner, G. S.; Buesseler, J. A., and Alfano, J. E.: Adrenocorticotropic Hormone (ACTH) and Cortisone in Ophthalmology, A. M. A. Arch. Ophth. **45**:301 (March) 1951.

3. Steffensen, E. H.; Wishbow, A. J.; Nagle, F. O.; Smith, R. W., and Whitney, E. L.: Topical Cortisone in the Treatment of Anterior Segment Eye Disease, Am. J. Ophth. **34**:345, 1951.

4. Leopold, I. H.; Purnell, J. E.; Cannon, E. J.; Steinmetz, C. G., and McDonald, P. R.: Local and Systemic Cortisone in Ocular Disease, Am. J. Ophth. **34**:361, 1951.

5. Hallett, J. W.; Leopold, I. H.; Vogel, A. W.; Cannon, E. J., and Steinmetz, C. G.: Treatment of Experimental Herpes Simplex Keratitis in the Rabbit, A. M. A. Arch. Ophth. **46**:33 (July) 1951.

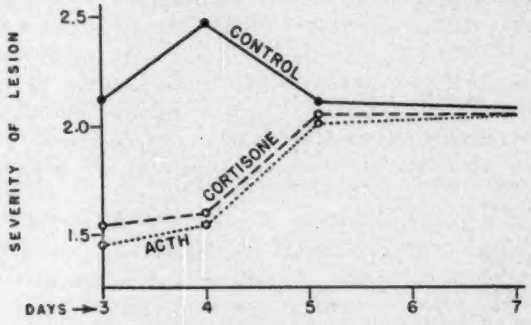
6. Loosli, C. G.; Hull, R. B.; Berlin, B. S., and Alexander, E. R.: Influence of ACTH on the Course of Experimental Influenza Virus Type A Infection, Proc. Central Soc. Clin. Research **23**:64, 1950.

virus-Type-A infections. Kilbourne and Horsfall [7] reported that the concentration of influenza or mumps viruses attained in developing hens' eggs injected with cortisone exceeded that in similarly infected control eggs by a significant amount. Shwartzman [8] claimed that intramuscular injection of cortisone in hamsters, which naturally have considerable immunity to poliomyelitis, transforms a negligible illness into a rapid, violent, and uniformly fatal disease; corticotrophin alone, however, did not affect the course of the poliomyelitis.

In an effort to shed more light on the effect of these substances on virus diseases in general, and herpes simplex in particular, we decided to supplement our unsuccessful experiments on local cortisone treatment in herpes simplex infections of the rabbit cornea with further experiments utilizing systemic cortisone and corticotrophin therapy.

METHOD

Twenty-four normal adult chinchilla and albino rabbits had each cornea anesthetized with a drop of 0.5% tetracaine-hydrochloride solution. Three linear, superficial, vertical incisions were



Influence of systemic administration of cortisone acetate and corticotrophin on experimental herpes-simplex keratitis in the rabbit.

then made through the corneal epithelium with a Graefe knife. A drop of macerated chorioallantoic membrane from a chick embryo infected with herpes simplex virus of the HS Rockefeller Institute strain was then instilled into the inferior cul-de-sac of each eye, and the lids were gently massaged over the cornea.

Starting immediately after corneal inoculation, eight rabbits were given 9 mg. of cortisone acetate daily intramuscularly in divided doses for four days, and then in a single dose for the next three days. Another group of eight rabbits received identical treatment with corticotrophin. The third group of eight rabbits constituted the untreated controls.

From the third day on, the corneas were stained with 2% fluorescein-sodium solution and counterstained with 2% methylene blue. Under suitable magnification, the lesions were observed and graded as to severity from 1 to 4, in accordance with the arbitrary system previously described.[5]

7. Kilbourne, E. D., and Horsfall, F. L., Jr.: Increased Virus in Eggs Injected with Cortisone, Proc. Soc. Exper. Biol. & Med. **76**:116, 1951.

8. Shwartzman, G.: Poliomyelitis Heightened by Cortisone, Proc. Soc. Exper. Biol. & Med. **75**:835, 1950.

RESULTS

Although there was a wide spread in the severity of the lesions in any one group, the average degree of severity for the 16 eyes in each group was computed for each day from the third to the seventh, inclusive. As the accompanying graph demonstrates, there was no difference between the cortisone and the corticotrophin (ACTH)-treated animals. The controls were more severely involved up to the fifth day. From then on the treated and the control groups presented practically the same degree of involvement.

It may be concluded that both corticotrophin and cortisone systemically administered have an identical retarding effect, up to the fifth day, on experimental herpes-simplex infections of the rabbit cornea. This is in contrast with the lack of benefit, or even deleterious effect, exhibited by local cortisone therapy.

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INDUCED OCULAR TUBERCULOSIS

Report of a Probable Case

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IN 1908, after 15 yr. of careful research, Calmette and Guérin produced a completely attenuated suspension of living tubercle bacilli, which was named BCG.[1] This suspension, when injected as a vaccine, produced a relative immunity which would give a certain degree of resistance to virulent tubercle bacilli. However, if an acquired resistance to virulent tubercle bacilli could be combined with a BCG vaccination, the immunity following the BCG vaccination could be converted into a more fixed immunity.[2]

The original BCG vaccine was so attenuated that it could not in itself cause progressive disease in experimental animals.[3] The cultures have been handed down from the original production, and, so far as is known, all the strains of the vaccine now in use have been derived from the attenuated culture produced by Calmette and Guérin.

Gradually, over the years, the value of BCG as a preventive of tuberculosis became more apparent. In 1921 Calmette permitted the BCG vaccination of newborn infants and advocated that his clear-cut results with animal immunization warranted an uncontrolled mass vaccination in France of all newborn infants.[4] On this recommendation, in France alone, 210,000 infants had been vaccinated with BCG [5] by 1930.

Vaccination became extremely popular in Scandinavia during World War II, and since Dec. 8, 1941, it has been extended in Denmark and Sweden to tuberculin-negative reactors over 14 yr. of age on a voluntary basis. No tuberculin-negative student nurse or medical student is admitted to nursing schools or medical faculties unless she or he is willing to become vaccinated with BCG.

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1. Calmette, A., and Guérin, C.: Contribution à l'étude de la vaccination des bovidés contre la tuberculose par les voies digestives, Ann. Inst. Pasteur **21**:525, 1907; Nouvelle contribution à l'étude de la vaccination des bovidés contre la tuberculose, ibid. **22**:689, 1908.

2. Calmette, A.: Guérin, C., and others: Essais d'immunisation contre l'infection tuberculeuse, Bull. Acad. méd. **91**:787, 1924.

3. Dubos, R. J.: Immunological Aspects of BCG Vaccine, editorial, Am. Rev. Tuberc. **60**:670, 1949.

4. Calmette, A.; Guérin, C., and others: Essai de prémunition par le BCG contre l'infection tuberculeuse de l'homme et des animaux, Bull. Acad. méd. **93**:78, 1925; Presse méd. **33**:825, 1925.

5. Broggers, A. W.: Septième Conférence de l'Union Internationale Contre la Tuberculose, Boktrykkeri, Oslo, 1931.

The BCG vaccination program is already in progress throughout New York State.[6] During 1947 more than 1,500 tuberculin-negative nurses, medical students, and hospital and sanatorium employees were vaccinated with BCG by the multiple-puncture method. In Pennsylvania approximately 8,000 medical students and nurses have been vaccinated during the past three years. All dental students in Pennsylvania are being vaccinated at the present time.[7]

The BCG vaccine is prepared by separating the culture (of desired virulence) from the synthetic medium by filtration. The bacillary mass is then suspended in dilute Sautan's medium or in buffered isotonic sodium chloride solution. The resulting vaccine is best used within five days, but can be used up to 40 days.

At present BCG vaccine is the only known immunizing agent which protects to a certain degree against tuberculosis. It can be of benefit only to persons who have not been infected with the tubercle bacillus. BCG has no value in the treatment of tuberculosis.

COMPLICATIONS OF BCG VACCINATION

There is general agreement on two points: (1) BCG vaccination is safe; (2) it seems to convert nonreactors to tuberculin to reactors.[8] Tytler[9] stated the belief that the harmlessness of BCG was established beyond doubt in that its practical use was exceeded only by a few proved methods of immunization, such as those of smallpox, diphtheria, and the enteric infections. Sweany,[10] as late as 1948, stated that beyond doubt BCG is harmless and that the only unfavorable feature is the danger of contamination or of mixing strains. Such an accident occurred in the Lübeck tragedy of 1930, in which, of 252 infants receiving accidentally an injection of a virulent human strain of tubercle bacilli, 72 died of tuberculosis.[11]

There are reported, however, some complications resulting from the vaccination, either by the multiple-puncture method or by subcutaneous injections. In practically every instance the vaccine induces a small ulcer when injected intradermally. This ulcer is followed by a superficial scar, the sizes ranging from 5 to 10 mm.[7] It is not unusual for the site to drain from four to six months. This will vary considerably in different persons.[12] The ulceration is not due to the growth of organisms, but is the result of substances from the bacilli which are injected into the skin. Ulceration may occur with dead bacilli.[7]

Vaccination with BCG by the subcutaneous route is known to be followed sometimes by the formation of nodules and fistulous abscesses. Blanch and associates[13] reported 12 cases of lymph-node enlargement, in two of which the mediastinal,

6. Birkhaug, K.: BCG and the Newer Epidemiology of Tuberculosis, Bull. New York Acad. Med. **24**:411, 1948.

7. Aronson, J. D.: Personal communication to the author.

8. Minutes of the Council, New York J. Med. **47**:2731, 1947.

9. Tytler, W. H.: Memorandum on BCG: A British Review, Lancet **2**:138, 1946.

10. Sweany, H. C.: Seminar on BCG: Introductory Remarks, Am. Rev. Tuberc. **57**:95, 1948.

11. Epilog zur Lübeck-Katastrophe, Med. Welt **5**:113, 1931.

12. Dubos, R. J.; Fenner, F., and Pierce, C. H.: Properties of a Culture of BCG Grown in Liquid Media Containing 'Tween 80 and the Filtrate of Heated Serum, Am. Rev. Tuberc. **61**:66, 1950.

13. Cantonnet Blanch, P.; Cantonnet Blanch, H., and Perez Scremini, A.: Las complicaciones ganglionares supuradas de la vacunación BCG Rosenthal, Rev. tuberc. Uruguay **16**:85, 1948.

inguinal, and supraclavicular nodes were enlarged. In Russia, a study was made of lymphadenitis among 2,700 infants given injections of BCG, 2.5% of whom showed some adenopathy.[14] This adenopathy was concluded to be benign.

In 1948 Le Melletier[15] described the case of a 2-wk.-old infant who was vaccinated with 0.04 mg. of BCG in the right axilla. The reaction to the tuberculin test was negative for three months, so revaccination was carried out in the left axilla. The reaction to tuberculin became positive after the second injection. Both sites of injection then broke down into cold-abscess-draining sites, and the bacteria of BCG cultures were obtained from each after three months. This case reveals that the bacteria remained alive in the infant's body at least that length of time. This partially proves the statement of Myers[16] that, although there is no immediate danger from the inoculation, no one has as yet proved that the organisms may not survive in the bodies of some persons for many years or decades.

Many cases of systemic, as well as ocular, reaction have been reported after the injection of tuberculin for the ordinary skin tests. Weizenblatt[17] reported a case of bilateral cyclitis and neuroretinitis which began five or six days after injection of 0.1 mg. of old tuberculin U. S. P. as a routine test on college students. Muncaster and Allen[18] reported a case of bilateral uveitis and retinal periarteritis which began several hours after the second dose of purified protein derivative of tuberculin U. S. P. In this case hospitalization was required for five weeks. The skin sites of each injection flared up initially, along with the eyes. There was essentially no resulting permanent loss of vision in either case.

In this respect, Woods[19] reported in 1943 that there was no relation between the degree of ocular sensitivity and the degree of cutaneous sensitivity. This is important in judging any ocular complication that might arise after any of these tuberculin injections. In immune-allergic reactions, such as could come after an injection of purified protein derivative of tuberculin U. S. P. or old tuberculin or in a combination of attenuated local infection, as in BCG vaccination, and an immune-allergic reaction unpredictable complications are possible.

The following case is presented as illustrating a possible complication of these injections; after a careful search of the literature, no similar case could be found related to BCG vaccination.

REPORT OF CASE

Miss A. A., a student nurse, aged 20, white, was referred to me by another oculist for consultation and treatment on May 4, 1949. Her complaint at that time was "sore and red eyes." This began about March 15. The condition had been intermittent, but had become progressively worse since the onset, although not severe enough to require medical attention.

The past medical history was taken and could be checked against the records of previous hospital admissions, since the patient was beginning her third year of training. Her past general

14. Martin, L. E.: BCG: A Review, Canad. M. A. J. **59**:467, 1948.

15. Le Melletier, J.: Petits abscès cutanés après revaccination par le BCG au siège d'une scarfication récent et d'une injection sous-cutanée antérieure, Rev. tuberc. **12**:235, 1948.

16. Myers, J. A.: BCG Vaccination, Am. Rev. Tuberc. **57**:107, 1948.

17. Weizenblatt, S.: Allergic Ocular Reaction to the Tuberculin Test, Arch. Ophth. **41**:436 (April) 1949.

18. Muncaster, S. B., and Allen, H. E.: Bilateral Uveitis and Retinal Periarteritis as a Focal Reaction to the Tuberculin Test, Arch. Ophth. **21**:509 (March) 1939.

19. Woods, A. C.: The Influence of Sensitivity and Immunity on Ocular Tuberculosis, Pennsylvania M. J. **46**:1133, 1943.

health records revealed nothing unusual. In December 1947 she was hospitalized and treated for five days for acute pharyngitis. There had been two other admissions of one day each for constipation, in February and in December, 1948. A blood-cell count and urinalysis were done on each occasion, in addition to the routine physical examination by one of the staff.

The further history revealed that, as a voluntary routine measure, a first-strength (0.00002 mg.) intracutaneous injection of purified protein derivative of tuberculin U. S. P. was made on Nov. 29, 1948. The reaction to this test was negative, so that a second-strength (0.005 mg.) injection was given on Dec. 7. The reaction to this injection was also read as negative.

On Feb. 7, 1949, 99 student nurses and instructors were given 0.1 mg. of BCG vaccine in one arm and 0.1 mg. of old tuberculin in the other arm, again as a routine procedure, under the direction of the staff of Phipps Institute, Philadelphia. This patient received the old-tuberculin injection in the right arm, and the reaction was read as negative at the end of 48 hr. At that time she was told of the soreness and drainage at the site of BCG injection to be expected in the near future.

After the injection the patient was perfectly well until the ocular symptoms began. She continued to work, however, and had been on duty the day she reported for ocular examination.

Ocular examination on May 4 revealed uncorrectible vision of 20/50 in the right eye and of 20/40 in the left eye. The conjunctiva was moderately injected (Fig. 1). The cornea was grossly clear. The anterior chamber was very slightly cloudy. The intraocular tension (Schiøtz) was 16 mm. in the right eye and 18 mm. in the left eye. Slit-lamp examination revealed considerable endothelial bedewing, a moderate number of cells, and a 2+ flare in the anterior chamber of each eye. There were early posterior synechias present.

The pupils were dilated with some difficulty after three instillations of 2% homatropine hydrobromide drops. Ophthalmoscopic examination revealed three hemorrhagic chorioretinal lesions near the macula of the right eye. No other definite pathologic condition could be seen in the posterior segment.

The patient was admitted to the hospital with an ocular diagnosis of acute uveitis anterior and posterior in the right eye and acute uveitis anterior in the left eye.

General physical examination on admission showed a normal condition except for a draining sinus on the left arm with surrounding inflammatory reaction, the entire diameter of the area being about 2 cm. (Fig. 2). This was the site of the previous BCG injection. There was no particular surrounding tenderness, and no axillary, cervical, or inguinal adenopathy was present. The temporary scab over this area was removed, and smear, culture, and guinea-pig inoculation of the material were made for tubercle bacilli, with negative results.

The patient was hospitalized from the date of the first examination, on May 4, until June 28, 1949. During the first 10 days the iritis gradually subsided; at the same time, the vitreous of each eye became cloudier, and more hemorrhagic chorioretinal lesions became visible. On May 14, acute bilateral optic neuritis developed, with a 1 D. elevation in each eye and scattered hemorrhages over the papilla, which were more pronounced on the right side. On May 23 vision was reduced to 20/200 in the right eye and to ability to count fingers at 2 ft. (60 cm.) in the left eye. Intraocular tension remained normal.

Repeated medical examinations of the internal organs revealed essentially nothing significant. Neurological examination showed a normal status. General surgical examination of the abdomen revealed nothing abnormal.

Six complete blood counts were done, the lowest of which showed 3,500,000 red blood cells per cubic millimeter. The white-blood-cell counts ranged from 6,500 to 12,500 per cubic millimeter. Two cultures of urine were made; they yielded no organisms. The blood-chemistry on two determinations was normal. Spinal-fluid examination, including colloidal gold test, gave normal results. Serologic reactions of the blood were negative. Two cultures of the stool revealed the usual flora. Two x-ray plates of the chest were comparable to the patient's normal one on admission to training. Roentgenograms of the skull and teeth revealed nothing abnormal. Agglutination tests for paratyphoid, typhoid and undulant fever gave negative results.

On her admission, the oral temperature was normal, but daily fluctuations, up to 99.6 F., were usually present for the first three weeks. Then the daily rise became as high as 102 F., and the pulse rate ran up to 120 a minute.

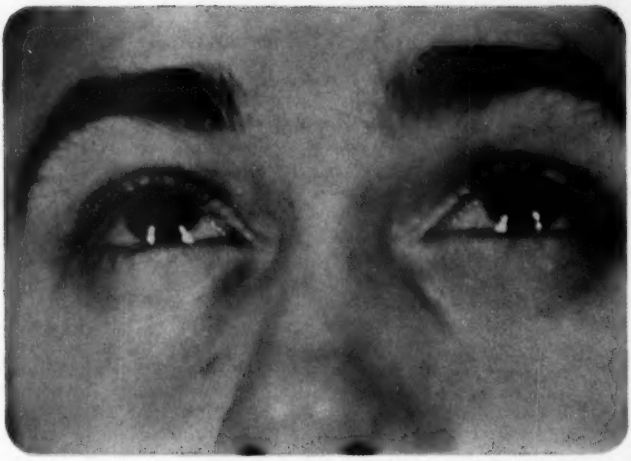

Fig. 1.—External view of patient's eyes on first visit, after three instillations of homatropine hydrobromide, 2%, in each eye, showing the rather mild degree of iritis present.

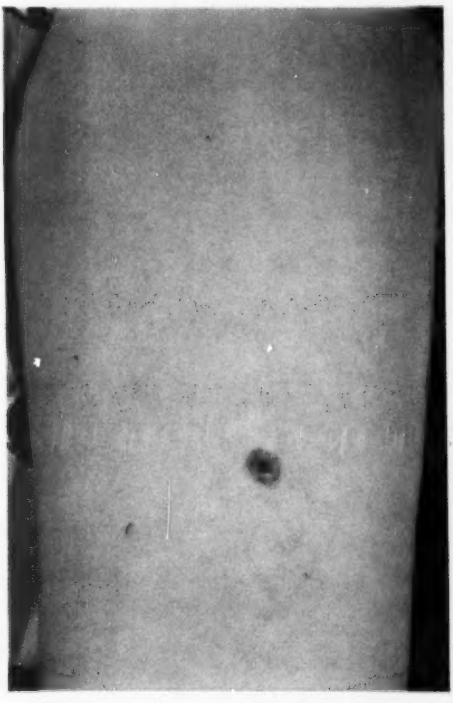

Fig. 2.—View of BCG reaction site on upper arm on first examination approximately three months after original injection, showing the draining sinus in the skin.

On June 2 vision was 6/200 in the right eye and was reduced to light perception in the left eye. No detail of the fundus was visible in the left eye. The vitreous had a red hemorrhagic glow. On June 14, the vitreous began to clear slightly, and for the first time there was a slight increase in vision. On June 23 diarrhea developed and persisted.

Ocular therapy consisted of the usual treatment of iritis, use of cycloplegics, and milk injections. Procaine penicillin was given daily for several weeks. Streptomycin was given on two occasions, 1.5 gm. daily for five days, until nausea, tinnitus, and dizziness forced discontinuation. Aureomycin was then given by mouth to replace the streptomycin. Tripelennamine ("pyribenzamine") hydrochloride, 150 mg., was given daily for the second week only.

The patient left the hospital on her own and her parent's volition, against the advice of all concerned, on June 28, with a temperature of 101 F.

On Aug. 2, 1949, the patient was brought to the hospital in a severe state of inanition, with abdominal pain, fever, and constant diarrhea. The red-blood-cell count was 2,590,000 per cubic millimeter, with a hemoglobin concentration of 55%. This count subsequently fell to 1,620,000, but gradually increased after five whole-blood transfusions. The white-cell-count varied from 5,500 to 16,200. The temperature was then running daily to 103 F. X-ray studies with barium revealed active ulcerative colitis. This was confirmed by proctoscopic examination as the usual acute ulcerative colitis by Dr. A. J. McAdams.

Ocular examination at the time of the second hospital visit revealed vision of 20/20 in the right eye and light perception only in the left eye. The eyes were externally grossly quiet. The pupils were well dilated from the local use of 1% atropine solution every second day. Ophthalmoscopic examination revealed the media to be fairly clear. The nerve heads showed slight secondary atrophy, and multiple chorioretinal scars were visible, with some retinitis proliferans. The intraocular tension (Schiøtz) was 18 mm. in the right eye and 37 mm. in the left eye. Use of atropine was stopped, and the patient was placed on a regimen of a drop of 1% pilocarpine nitrate in the left eye only three times a day. The tension promptly fell to normal in this eye.

After discharge, on Sept. 3, 1949, the patient was sufficiently cooperative that she could be followed routinely. In December, 1949, a low-grade interstitial keratitis developed in the left eye; this gradually healed after two months but left a deep, diffuse stromal scar in the cornea inferiorly.

In February, 1950, the patient was admitted with atypical interstitial keratitis in the right eye, which on the surface suggested a dendritic configuration. This hospitalization lasted eight days, and the keratitis healed, leaving a small, deep scar.

In March, 1950, vision in the right eye was 20/50, and in the left eye it remained limited to light perception. Without any miotics, the tension had remained within normal limits.

In November, 1950, the patient had a recurrence of diarrhea, lost 12 lb. (5.4 kg.), and was readmitted to the hospital. Proctoscopic examination again revealed the rectal ulcers, which this time gave the clinical appearance of tuberculosis. Culture of scrapings from one ulcer yielded no tubercle bacilli. The patient did not desire a second proctoscopic examination. The ocular findings had not changed since March, 1950.

On Feb. 12, 1951, she was again admitted to the hospital, with an oral temperature of 99.4 F. and constant diarrhea. Her weight on this admission was 85 lb. (38.6 kg.).

The patient is continuing, with some difficulty, her nursing career, under constant medical surveillance and recurrent admissions to the hospital.

COMMENT

Two main questions arise in this case. The first is whether any cause other than the BCG vaccination could be found for the ocular involvement. Studies and subsequent follow-up observation suggest no other cause except the ulcerative colitis, which is probably also the result of injection, particularly since it manifested itself so long after the ocular changes. The second question is, How much of this is due to the BCG vaccination and how much to the injection of old tuberculin? Since the reaction to old tuberculin was negative and it, in turn, did not affect the sites of previous injection of purified protein derivative, this case certainly differs from

reported cases of ordinary immune-allergic reactions. In the latter, also, the ocular reaction occurred within a matter of days, instead of five weeks, as in this case.

The fact that there was no lymphadenopathy at any time could be ascribed to the fact that five weeks had elapsed since injection, when the patient was first seen, or to the rather selective ocular and intestinal involvement.

Aronson [7] has suggested that another injection of old tuberculin be given, in an effort to verify the findings, since it would cause another flare-up if it were related to the causative agent. He agrees with all of us that the risk involved would be very great, and further skin tests will not be done.

SUMMARY AND CONCLUSIONS

A case of diffuse bilateral ocular involvement, clinically tuberculous, probably induced by BCG itself or BCG in combination with injections of tuberculin, is presented.

Observations in this case cover the past two years, and the present visual loss is permanent, although further complications are possible.

The ultimate prognosis so far as life is concerned is as yet undetermined in this case.

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ANALYSIS OF METHODS FOR MEASURING DIPLOPIA FIELDS

Introduction of Device for Such Measurements

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ONE OF the most exciting chapters in the history of ophthalmology pertains to the work on motor anomalies of the eye. The student repeatedly meets the same names, which are like giant guide posts indicating and directing in logical steps the accumulating knowledge in this field. When one considers the limited technological equipment available to these men, and then realizes that the newest of our techniques were basically described before the beginning of the century, an appreciaiton for their stature is obtained. Certainly, the names of Alfred Graefe, Hirschberg, Mauthner, Bielschowsky, and Hering give Germany its share of giants. In like manner, England has produced its Maddox, and France, its Javal and Landolt, with his genius for establishing method. No less do the early great names stand out in this country: Savage,[1] Stevens, Duane, Howe, J. C. White and, now, Lancaster. As the result of the teachings of these masters, many new names will appear, and to a considerable degree men from this country, where in the past 20 yr. leadership in this field seems to have been established. Contributions have stemmed not only from ophthalmologists but also, and to a notable degree, from physiologists and mathematicians.

It may be categorically stated that if one understands the actions of the various extraocular muscles, one will have no great difficulty in evaluating the diplopia fields by any of a number of techniques. Since the middle of the 19th century, articles have appeared in the literature by various authors illustrating and teaching a particular method for the measurement of diplopia fields. It would appear that an analysis of these various methods is indicated so that the reader may decide which technique is more applicable under average circumstances. The importance of ease in carrying out a test, the duration of time required to make the test, and the comprehensibility of the test to the average patient and to the average physician, would then tend to make certain procedures preferable to others.

Since the actions of various extraocular muscles had for a long time been an enigma to the average ophthalmologist, many authors have attempted to make their tests such that a minimal working knowledge of the individual actions of the various muscles was required. One only had to follow a particular rote and then, analyzing these findings by a few simple rules, arrive at a proper diagnosis. Many of the men who devoted considerable amounts of their time to devising such tests were also pioneers in education of the ophthalmological world to a better working knowledge of the motor anomalies of the eye.

Accepted as the candidate's thesis for membership in the American Ophthalmological Society (1950).

1. Savage, G. C.: Ophthalmic Neuromyology: A Study of the Normal and Abnormal Actions of the Ocular Muscles from the Brain Side, Nashville, Tenn., A. R. Gray & Son, 1905.

It is fitting to state that the earliest works stressed the importance of observing the movements of the eye in order to identify the lagging muscles in cases of diplopia produced by paresis.

Mackenzie,[2a] in 1833, stated, in speaking of total third-nerve paralysis:

> If with the finger we lift the upper lid in such a case, and tell the patient to look to the ground we see that he attempts to do so but is utterly unable to accomplish his intention.

In the fourth volume of this book,[2b] published in 1854, he noted:

> . . . By passing the finger from side to side and upward and downward before the patient and desiring him to follow it with his eyes without moving his head, we detect which eye and which muscle is affected, even when the loss of power is very slight. In general it is the adductor or abductor of one or the other eye that is defective so that the organ cannot be completely inverted or everted. Thus diplopia is experienced in general when the patient endeavors to look straight before him and always when he tries to look in the direction opposite to the deviation of the affected eye. If the levator oculi for instance is palsied, diplopia occurs whenever the patient attempts to look upward. In cases of palsie of the fourth nerve or of the inferior rectus the rotary motion of the eye will be interfered with as may perhaps be detected by laying hold of the patient's head and bending it from shoulder to shoulder.

Carter,[3] in 1876, described the use of a colored glass and an elongated fixation testing object, such as a roll of white paper; thus one could see the inclination, as well as the doubling, of the images.

Soon it became apparent that evaluation of the character of the subjective diplopia field and proper identification of the image belonging to each eye was a more satisfactory method. No doubt the influence of accumulated knowledge and clinical information was largely responsible for the acceptance of this method. It set the background for the better understanding of diplopia. Thus, it appears that there came a shift in emphasis from that of evaluating the lag in the paretic muscle and then explaining the diplopia, to that of first interpreting the subjective diplopia and then identifying the involved muscle. Thus, the most popular methods of measuring diplopia fields became those employing subjective responses by observing the doubling of images and identifying the image of one or both eyes by the use of an appropriately colored filter. The simple red-glass test, in which the observer watches a moving light and describes the field of greatest diplopia, is perhaps the oldest, and still most widely used, method of testing. Hering's[4] law (1879) of equal and symmetrical movement of the two eyes did much to explain the difference between primary and secondary deviation.

RED-GLASS TESTS

Hirschberg [5] was one of the early investigators to measure the angle of deviation. He employed a wall painted in calibrated arc degrees, used a red glass, and plotted his findings in terms of separation of images for the various positions tested. He

2. Mackenzie, W.: (*a*) A Practical Treatise on the Diseases of the Eye, Boston, Carter Hendee & Co., 1833, p. 203; (*b*) Ed. 4, revised and enlarged, Philadelphia, Blanchard & Lea, 1854, p. 347.

3. Carter, R. B.: Practical Treatise of Diseases of the Eye, Philadelphia, H. C. Lea, 1876, p. 442.

4. Duke-Elder, W. S.: Text-Book of Ophthalmology, Vol. 1, St. Louis, C. V. Mosby Company, 1932, p. 602.

5. Hirschberg, J.: On the Quantitative Analysis of Diplopic Strabismus, Brit. M. J. **1**:5-6, 1881.

first worked at a 2-m. distance, then used 1 m. His assistant, Ohm, later indicated his findings in a similar manner (Fig. 16). Hirschberg noted that the method "is based upon the patient's subjective measurement of the reciprocal distances of the images. To render the results more apparent, some authors have introduced diagrams or divisions." A division into numbered squares is described in "Mr. Bader's [6] Text Book (1868)" and "a similar division of a wall into squares of 0.25 meters in length was adopted by Heiberg [7] of Christiana in 1872." Hirschberg in 1875 improved on these methods by using an angular division, rather than the longitudinal distance, an "innovation [that] is not a trifling one, as it may at first appear, but rather essential for our purposes, as in perimetry of the visual field." He commented on the method of testing for diplopia with prisms:

> By the use of prisms two sources of error are introduced. The first is a faulty position of the prism; the second and more important is the tendency to a fusion of the two images.

In experiments he conducted he showed that

> "at first the deviation is equal to 3°, after a few seconds 2° and shortly it becomes zero. That is to say, single vision is obtained by the tendency to the fusion of both of the images."

Landolt,[8] in his book published in 1879, made reference to the use of a red glass before one eye as being most important. (It is interesting to note how many of the leading ophthalmologists used the red-glass test in evaluation of diplopia purely as a qualitative method and ignored the quantitative aspects.) This simple test is referred to in most of the standard textbooks [9] up to recent years, where it appears that the differences described occur only in the method of depicting the nature of the diplopia. It is particularly a good test when one is dealing with a very intelligent patient, but it is good to the point that one will get a qualitative, rather than a quantitative, measurement.

But in 1879 Landolt [10] described a technique for measuring the actual amount of diplopia. He set up a tangent cross made up of tapes calculated at 5-degree intervals for a 3-m. range. A red glass was placed before one eye of the patient. The position of the red image indicated directly the angle of the squint by its position from the white image in distance of tapes. The patient's head was kept still, and the candle was moved in the different positions of gaze. At this point, another important reference was made by Landolt:

> We come now to an important method of correcting and at the same time measuring strabismus; that is the use of prisms. In looking through a prismatic glass we find that it produces an effect analogous to that of the deviation of the eye; that is to say it causes displacement of the objects looked at. It follows from this that employed in a contrary sense the prism would neutralize the diplopia caused by the deviation of the eye.

Thus, over 70 yr. ago two techniques were described not unlike that practiced, justifiably, by many ophthalmologists at this time, namely, (1) measuring the

6. Bader: Text Book, cited by Hirschberg.[5]

7. Heiberg: Ophthalmological Methods, cited by Hirschberg.[5]

8. Landolt, E.: A Manual of Examination of the Eyes: A Course of Lectures Delivered at the "École Pratique," translated by S. M. Burnett, Philadelphia, D. G. Brinton, 1879, pp. 59-61.

9. Noyes, H. D.: Text-Book of Diseases of the Eye, Baltimore, William Wood & Company, 1890, p. 139.

10. Landolt, E.: Untersuchungmethoden, in von Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, Ed. 2, Leipzig, W. Engelmann, 1904, Vol. 4, p. 737.

deviation by interpolating the linear deviation into degrees of separation of images; (2) neutralizing the diplopia with prisms.

In 1893 Ziegler [11] described the use of a scale calibrated in arc-degree intervals, containing both a vertical and a horizontal component, so that diplopia could be measured directly. This protractor-like device was used primarily for the measurement of the power of prisms (Fig. 1).

Maddox,[12] in his memorable work on "Tests and Studies of the Ocular Muscles," published in 1898, described the use of a tangent scale and his glass rod. A cross was constructed containing a space for a central light; arc degrees were then indicated on the cross, calibrated as a tangent scale for 5 m. The patient was seated 5 m. away, and readings were taken with the head in nine different attitudes. Unlike Landolt, who moved the test object, Maddox moved the head in order to get the effect of rotation of the eyes. A Maddox rod was held before one eye, and the patient indicated where the streak passed through the scale; the rod was

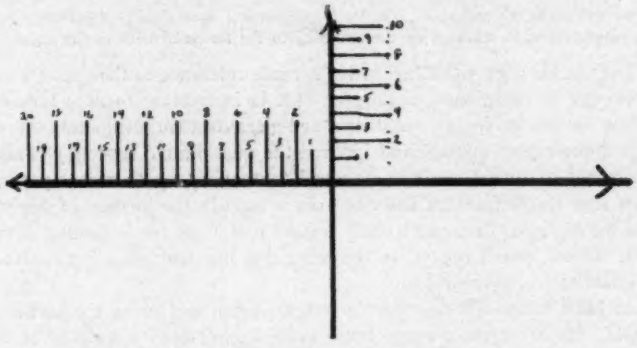

Fig. 1.—Ziegler's scale.

held appropriately to get the vertical deviation and was moved 90 degrees to get the horizontal deviation. Maddox stated:

> It is always a good plan, when time permits, to take as accurate measurements as we can of the diplopia in all nine areas of the motor field. By repeating such measurements from time to time an excellent idea of the progress of the case is formed and difficulties in the diagnosis may be cleared up.

Maddox also specified a particular way of indicating the degree of diplopia. He suggested that one make a square for the position of gaze that is to be depicted, place a dot in the center of this square to indicate the position of the true image, and then place a second dot to indicate the false image. One then draws a faint line horizontally and vertically to connect the two dots, indicating the degrees of displacement, and, whenever possible, draws the inclination of the rod if it is not seen as perfectly vertical or horizontal.

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11. Ziegler, S. L.: A Convenient Prism Scale, Ann. Ophth. & Otol. **2**:262-272, 1893. Howe, L.: The Muscles of the Eye: Pathology and Treatment, New York, G. P. Putnam's Sons, 1908, Vol. 2, p. 190.

12. Maddox, E. E.: Tests and Studies of the Ocular Muscles, Bristol, J. Wright & Co.; London, Simpkin [and others], 1898, pp. 288-289.

One of the greatest influences in diplopia-field measurements was that of Duane.[13] He advocated using the reverse (white) side of a curtain tangent screen; the other side (black) was turned toward the patient, who sat 30 in. (75 cm.) away, with his eyes opposite the center. A red glass was placed before the right eye; a light was then placed in six cardinal positions. The patient turned his eyes, not his head. The location of the images was then indicated with pins, thus noting the displacement in terms of degrees. On the white side of the curtain one could evaluate the diplopia. Duane[14] suggested indicating one eye (in diagraming the field) as a ring, the other eye as an X. The circles on the tangent screen were at intervals of 5 degrees, calibrated for a distance of 30 in.; diameters passed through the center of the circle at 15-degree intervals, and the chart was further subdivided into a checkerboard pattern of squares, each square being approximately 1 degree. In this way, it was easy to transfer the findings onto a reduced chart for record purposes.

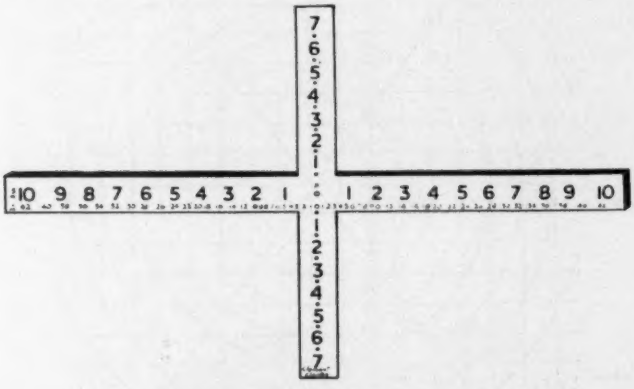

Fig. 2.—Maddox tangent scale (Lyle and Jackson).

Stevens,[15] in 1906, introduced a perimetric method of measuring the diplopia. A rider, which could be moved on the arm of the perimeter until it seemed to obstruct the image of the deviating eye, would be moved to such a position, and the arc degrees of diplopia could then be read directly; that is, the patient fixed the center of the perimeter and indicated the direction of the second image. Then the obstructing rider would be moved into this position, thus appearing to be where the second image was. Stevens condemned this method himself on the basis that

13. Duane, A.: New Classification of Motor Anomalies of the Eye Based upon Physiologic Principles, Together with Their Symptoms, Diagnosis and Treatment, [the Prize Essay of the Alumni Association of the College of Physicians and Surgeons, New York, for 1896], Ann. Ophth. & Otol. **5**:1002, 1896; New York, J. H. Vail & Co., 1897.

14. Fuchs, E.: Textbook of Ophthalmology, translated and edited by A. Duane, Ed. 8, Philadelphia, J. B. Lippincott Company, 1924, p. 301.

15. Stevens, G. T.: Motor Apparatus of the Eye, Philadelphia, F. A. Davis Company, 1906, p. 470.

it was "clumsy, rather troublesome and above all not of great utility." He also noted that prisms when "properly used gave the most uniform result, although no measurements are entirely satisfactory, since they may vary from hour to hour." While, therefore, making his quantitative perimetric method not of greatest value, and paying a somewhat reserved tribute to prisms, he gave his vote for his tropometer. He stated:

. . . this offered a perfect means of measuring the rotating ability of an affected muscle. By this means one could estimate the *real extent* of the disability.

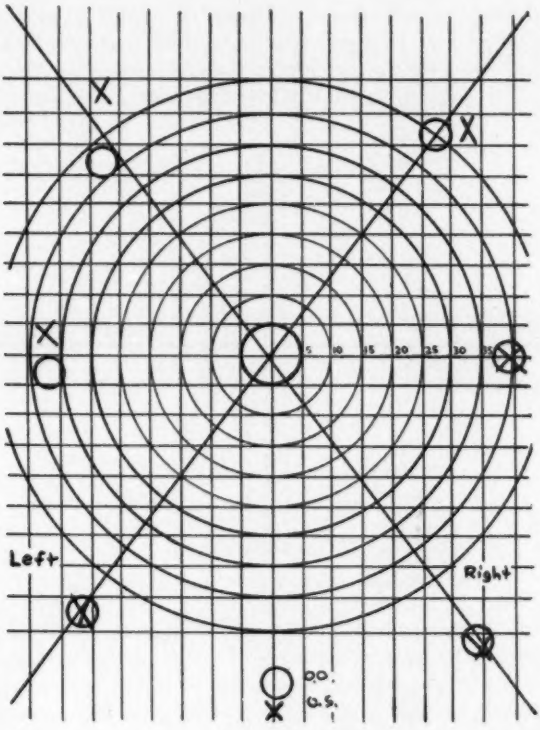

Fig. 3.—Duane's screen. Squares in actuality are 1 degree each. Findings in a case of paresis of the left superior rectus: right eye, O; left eye, X.

The tropometer (Fig. 4), presented in 1894, was an instrument designed to measure the various rotations of the eye. It was made of a telescope with a 45-degree prism or mirror at its objective end, so as to reflect at right angles. The images of the eye were seen along with appropriate readings of the scale. As the eye was made to move, the deviation could be expressed quantitatively by reference to the scale. He was the last of the great contributors to give preference to a measure of rotation of an eye as the best index of the presence of a paresis.

It is interesting to note that Weeks,[16] several years later, made this mention:

> Instruments for measuring limitation of movement are not as reliable as the measurement of the field of binocular vision with the candle which gives the amount of diplopia in prisms in different directions of gaze.

Tiffany,[17] in 1902, used the light and the red glass at a distance of 15 to 20 ft. (4.8 to 6 m.) from the patient. Even at this far distance, he permitted no movement of the head, but had the patient follow his light, denoting the position of the images and separation. Thus, he depended on a qualitative, rather than a quantitative, analysis; on the other hand, he measured the degree of strabismus with the perimeter. Despite the valuable contributions of Landolt,[18] Maddox,[12] and Duane,[13] many of the authors of the day continued to describe diplopia-field testing in a qualitative, rather than a quantitative way. Nettleship[19] used a candle and a red glass at 6 ft. (180 cm.), moved the light, and simply recommended a different

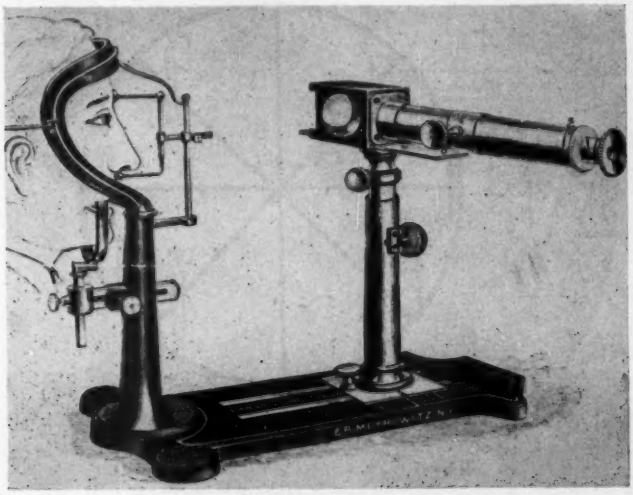

Fig. 4.—The tropometer.

way of recording the findings. The diplopia and the relative position of the images were obtained by questioning and were recorded on a chart, such as that shown in Figure 5.

Edward Jackson,[20] in 1900, described the red glass and a flame as a test for diplopia and said that one "might use a handle of the reflecting ophthalmoscope to arrive at the decision as to whether there is a torsion type of deviation."

16. Weeks, J. E.: A Treatise on Diseases of the Eye, Philadelphia, Lea & Febiger, 1910, pp. 725, 738 and 739.

17. Tiffany, F. B.: Anomalies and Diseases of the Eye, Kansas City, Mo., Hudson Kimberly Pub. Co., 1902, p. 543.

18. Landolt,[10] p. 659; p. 737; footnote 8.

19. Nettleship, E.: Diseases of the Eye, Philadelphia, Lea Bros. & Co., 1900, p. 380.

20. Jackson, E.: Essentials of Refraction and of Diseases of the Eye, with a Consideration of Ocular Injuries and the Ocular Symptoms of General Diseases, Philadelphia, W. B. Saunders Company, 1900, pp. 207-208.

De Schweinitz [21] noted Duane's strong influence; yet this important person gave no indication of the importance of quantitative measurements and simply referred to the red glass and the candle. Parsons [22] deviated a bit by suggesting that the lighted candle be kept 4 ft. (120 cm.) from the patient and the patient move the head, rather than the light; and he indicated a slightly different way of recording on a chart. All of the recordings up to this period are so that the right side appears on the right side of the chart and the left side on the left side of the chart.

Bielschowsky [23] fully established his high position as an authority in the field of motor anomalies in 1912, when he was made professor of ophthalmology and director of the eye clinic of the University of Marburg. His influence on American ophthalmologists was tremendous, as a result of his many lectures and demon-

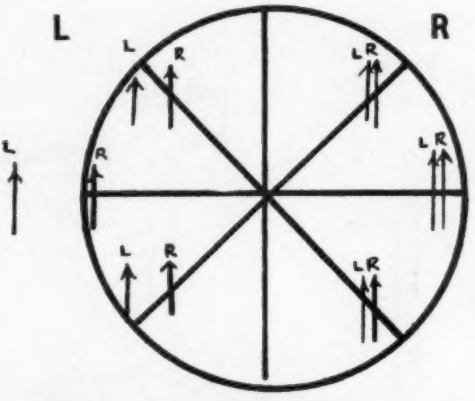

Fig. 5.—Representation of diplopia field in a case of paresis of the left lateral rectus (Nettleship).

strations on ocular motility. Many of his audiences were persons with inadequate training in the basic knowledge of eye movements, and he earnestly did much to bring enlightenment. His great contributions are of increasing significance, since they paved the way for renewed interest in such related fields as orthoptics, and made possible a deeper appreciation of the work of our own ophthalmologists, such as Duane, Stevens, White, Savage,[1] Howe, and Lancaster. He no doubt inspired many to work in this field whose names we shall soon learn to remember.

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21. de Schweinitz, G. E.: Diseases of the Eye, a Handbook of Ophthalmic Practice for Students and Practitioners, Ed. 10, Philadelphia, W. B. Saunders Company, 1924, p. 616.

22. Parsons, J. H.: Diseases of the Eye, Ed. 9, New York, The Macmillan Company, 1938, p. 536.

23. Lancaster, W. B.: Foreword in Bielschowsky, O.: Lectures on Motor Anomalies, Hanover, N. H., Dartmouth College Publications, 1940.

Bielschowsky [24] recommended and used the red glass [25] and the Maddox tangent cross in measuring diplopia fields. He advocated turning the patient's head to get the various positions of gaze that he wished to test:

> The patient is seated, his head in the normal position facing the light in the center of the tangent scale at a distance of 2.5 m. on a level with his eyes in the median plane of his head. A dark red glass is put first in front of the normal eye, then in front of the paretic eye. The red glass should be so dark that the eye behind it sees nothing but the colored light. Instead of moving the light fixated by the patient, the doctor turns the patient's head so that he has to move the eyes in the opposite direction in order to maintain fixation. The passive rotation of the patient's head to induce the various ocular movements instead of shifting the light through the whole field of fixation has the advantage that the doctor is able to control the position of the patient's head and therefore his eyes during the whole examination.

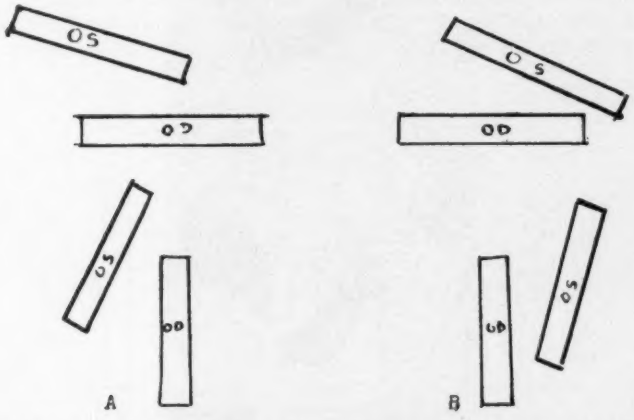

Fig. 6.—Paresis of the right trochlear nerve (Bielschowsky), right eye fixing. The double images in *A* indicate an associated homonymous diplopia, whereas in *B* the horizontal associated diplopia is crossed. Though the relative extortion is the same in both cases, the two images of a vertical object will converge or diverge above, whereas the two images of a horizontal object in such a case will always converge to the affected side.

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24. Bielschowsky, A.: Lectures on Motor Anomalies: Signs and Symptoms of Heterophoria, Am. J. Ophth. **21**:1219-1229, 1938.

25. In the choice of the red glass, Bielschowsky [24] states: "The red glass must be so dark that the eye behind it does not see anything but the red light; otherwise the patient would see double images of the whole tangent scale and be unable to make exact statements." Scobee (The Oculorotary Muscles, St. Louis, C. V. Mosby Company, 1947, p. 271) noted that the red glass should be "no darker than is necessary to impart a reddish tinge to the white light." A very dark lens tends to disrupt fusion and uncovers a heterophoria which may influence the findings.

I feel that, assuming there were a constant critical dividing line between a heterophoria and a heterotropia, the use of a graded colored glass to determine this point would still be subject to many variables, such as intensity of illumination, distance, and technique. It is evident that Bielschowsky's method of testing would become confusing to the patient if he were to visualize a second Maddox cross through a light-red glass. Certainly, it is not usual to have an anatomic heterophoria of such an order as to disguise the nature of a paresis when the diplopia is evaluated in the field of the paretic muscle.

Thus the patient is given opportunity to note the position of the red image in relation to the tangent scale on the cross. The patient may then indicate this position by describing it in terms of numbers to the right or the left of the light, or, what has proved to be more practical, by going up to the chart and pointing. In this way, the diplopia is measured quantitatively in the various positions of gaze.

Fig. 7.—Head-tilting test (Bielschowsky).

We shall consider the right eye in a case of paralysis of the right trochlear nerve: In tipping the head to the right, there is a compensatory vestibular innervation to intort the right eye and to extort the left eye (levocycloversion). The intorsion of the right eye would be brought about normally by the additive action of the superior oblique and the superior rectus muscle. The respective elevating and depressing actions of these two muscles would, however, be neutralized. In the absence of the depressing function of the paretic superior oblique, there would be no resistance to the elevation produced by the superior rectus, and thus the eye would assume an elevated position, producing doubling of the line.

Since the eye behind the red glass is the fixing eye, the primary deviation is measured when the filter is held before the nonparetic eye, and the secondary deviation is indicated when the glass is held before the paretic eye.

In the cases in which one wishes to evaluate fully the obliquity of double images, Bielschowsky [26] made a very valuable contribution. He contended that one should avoid the use of a vertical test object, such as the conventional candle. (The same reasoning, of course, applies to the bar light of Anderson [27] or to a roll of paper [3] held vertically.) Torsion of the vertical meridians of double images will appear different according to whether there is, besides the vertical deviation, a homonymous or a crossed diplopia. Thus, in spite of the same paresis, the two images will diverge or converge upward, owing to the influence of the horizontal element of the diplopia. This can be avoided by using a horizontal object.

The head-tilting test [28] is a qualitative test which brings out effectively the influence of the position [29] of the head on the diplopia. Bielschowsky used a simple apparatus based on the principle of Helmholtz' *Visierzeichen*. The relation of the patient's head to the object of his attention is fixed by having him bite on a small plate at the end of a rod, to which is connected 30 in. (75 cm.) away a piece of white cardboard containing a black horizontal line. Thus, the tilt of the patient's

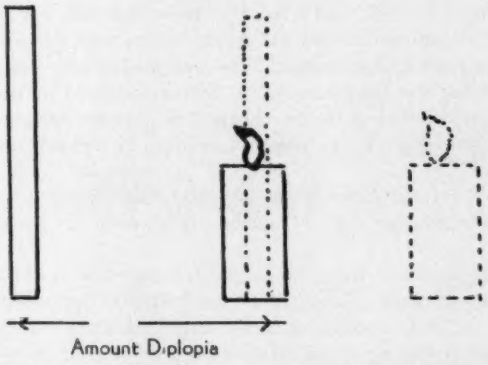

Fig. 8.—Double images of two objects (Kestenbaum).

head will determine the tilt of the cardboard; a tubular hand support is supplied to hold this rod, without interfering with its rotation. Doubling of the horizontal line viewed will occur if the head is tipped one way and will be less or absent if the head is tipped in the other direction.

Kestenbaum,[30] in 1946, described an interesting way of measuring the diplopia without the use of a colored glass. In addition to a light, a second object, e. g., a pencil, is held before the eyes (Fig. 8). Thus the patient sees two objects doubled:

26. Bielschowsky, A.: Lectures on Motor Anomalies: Paralyses; General Symptomatology, Am. J. Ophth. **22**:279-288, 1939.

27. Duke-Elder, W. S.: Diagram Showing Bar Light Used by Anderson, in Text-Book of Ophthalmology, Vol. 4, C. V. Mosby Company, 1949, p. 3875.

28. Bielschowsky, A.: Lectures on Motor Anomalies: Paralysis of Individual Eye Muscles; Abducens-Nerve Paralysis, Am. J. Ophth. **22**:357-367, 1939.

29. Hoffman, F. B., and Bielschowsky, A.: Die Verwertung der Kopfneigung zur Diagnostik von Augenmuskellähmungen, von Graefes Arch. Ophth. **51**:174, 1900.

30. Kestenbaum, A.: Clinical Methods of Neuro-Ophthalmological Examinations, Grune & Stratton, Inc., 1946, p. 156.

two images of the light; two images of the pencil. The pencil is now moved until one of its images is superimposed on the image of the light; the distance between the pencil and the light, therefore, is the measure of the diplopia and can be estimated by the examiner, or he may measure it directly. The test may be carried out in the various positions of gaze; thus, without equipment other than a small ruler, the examination can be made in an effective way. Kestenbaum also suggested what he calls the "Fixation-shift" test, in which the patient is asked to estimate the distance between the two images. In order to do this, he looks from one image to the other, making a number of changes of fixation; the examiner notes the movement the eye makes (for each millimeter, about 5 degrees would be the amount estimated). It would seem that this technique might be better in theory than in actual practice. The author also suggests the use of a screen which is not calibrated, on which the patient indicates the position of the second image and the examiner measures it directly with a ruler.

THE RED-GREEN TEST

Ewald Hering,[31] in 1899, used a red filter before the right eye and a green filter before the left eye and moved red and green targets seen monocularly with each eye until they appeared superimposed. He also used a faint red-and-green light. Thus, in first using the red-green test to demonstrate and measure the angle of squint by the displacement of the two images, he gave the background of all such methods, viz., by using (1) a darkened room, (2) red-green filters and (3) red-green lights.

Duke-Elder [32] made a statement to the effect that the red-green test is a type of cover test in which one eye is "functionally covered" as far as the other eye is concerned.

An ingenious deviation from the simple red-glass test is this use of complementary red-green targets. Essentially,[33] the difference between this type of test and the simple red-glass test is that in the latter there are double images of one light—the patient is looking at one fixation object, i. e., one light which is visible to both eyes. The red glass simply helps to identify the image of one eye. Since the two eyes are not looking directly at the same object, the image will fall on the fovea of one eye and will be in an extrafoveal position in the other eye; thus, the image of the eye in the extrafoveal position will be projected according to the distance from the fovea. A person who has an esotropia, while in actuality the eyes converge toward each other, will project images that are uncrossed; in a similar way, a person who has an exotropia, in which the eyes diverge from each other, will project as though the eyes were crossed; i. e., crossed diplopia will occur. Since the optical understanding of this is not nearly as important as the clinical report of the patient, it has not been hard for the ophthalmologist to appreciate that a crossed diplopia means a divergence of the visual axes and an uncrossed diplopia means a convergence of the visual axes. Thus, it has become a sort of

31. Hering, E.: Über die anormale Lokalisation der Netzhautbilder bei Strabismus alternans, Deutches Arch. klin. Med. **64**:23, 1899.

32. Duke-Elder, W. S.: Text-Book of Ophthalmology, Vol. 4, St. Louis, C. V. Mosby Company, 1949, p. 3875.

33. Burian, H. M.: Sensorial Retinal Relationship in Concomitant Strabismus, Arch. Ophth. **37**:336-368 (March) 1947.

second nature to accept this finding. Now, in the matter in which complementary red-green filters are used before the two eyes, two lights, or two test objects, are used. One is red, and is seen only by the red-covered eye; the other is green, and is seen only by the green-covered eye. Since the person will see the respective test object with the fovea of the appropriate eye, the images will be projected along the true direction of the eye. Thus, a person who has convergent strabismus, in looking at red-and-green, similarly located targets, will project the image of each eye as though it were convergent or crossed in relation to that of the other, whereas in divergent strabismus the images will appear in the form of a homonymous relationship. This is in keeping with the direction of the eyes; but, because it is different and diametrically opposed to what one has been accustomed to in the use of the other types of muscle tests, for example, the Maddox-

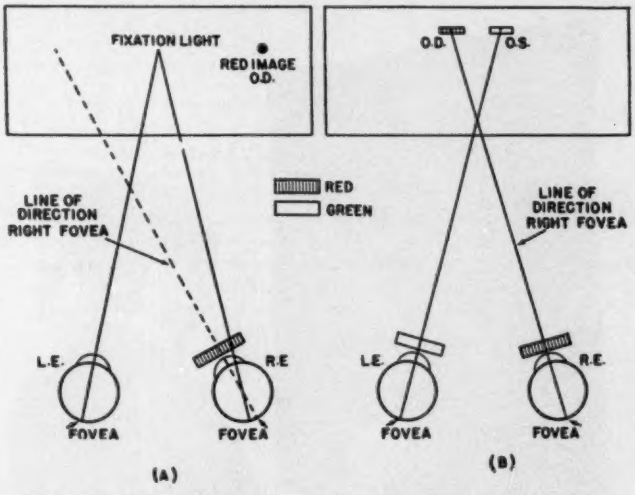

Fig. 9 (Burian).—*A*, the double-image test; *B*, the red-green test (Lancaster). In *A*, the fixation light is imaged on the fovea of the fixating eye and a peripheral element of the deviated eye; result, uncrossed diplopia. In *B*, each test line is imaged on the fovea of the eye with which it is seen; result, crossed diplopia.

rod test, in which the reverse positioning of images occurs, confusion sometimes arises. Nevertheless, if the situation is understood, a valuable test is available for such studies.

By the turn of the century the red-green test had found many uses in ophthalmology, particularly in the malingering test (FRIEND) and in the Worth [34] four-dot test for measuring the status of binocular vision. An American ophthalmologist, Charles Williams,[35] constructed a cross made of four hollow tubes arranged with incandescent bulbs and red filters. In front of this cross he arranged a series of

34. Worth, C.: Squint: Its Causes, Pathology and Treatment, Philadelphia, The Blakiston Company, 1903, pp. 14-15.

35. Williams, C. H.: A New Instrument for Testing the Position of the Axes of the Eyes, Tr. Am. Ophth. Soc. **9**:388, 1901.

illuminated green numbers, each separated by the distance of an angle of deviation calculated for 20 ft. The patient wore complementary red-green glass goggles, and the deviation for the primary position of gaze could be read directly. By controlling the switches so as to illuminate first the vertical arm of the cross, which was red, and the horizontal green numbers, one measured the horizontal deviation. Then, with similar switches, the horizontal red line was made to appear along with

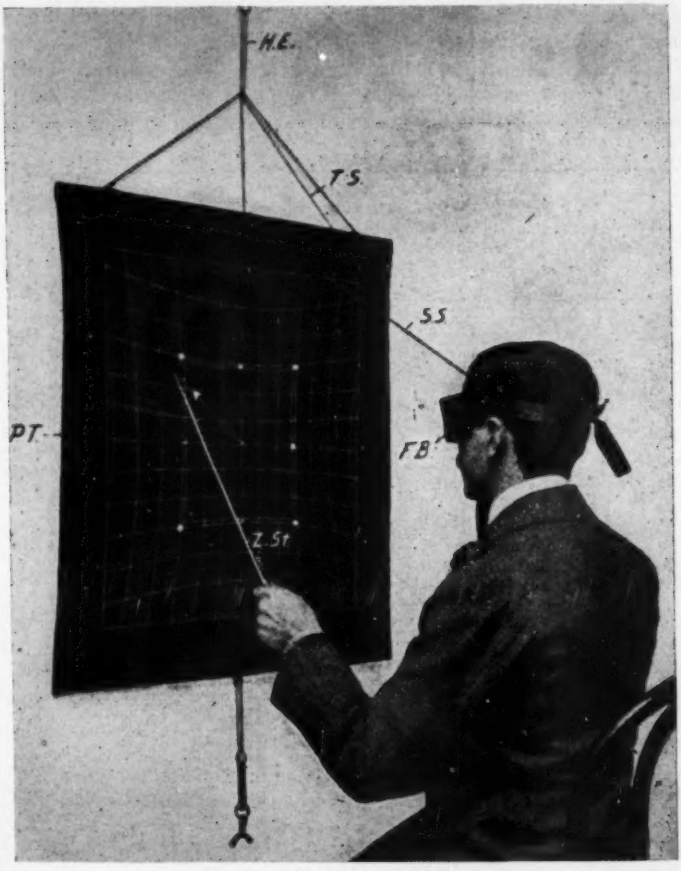

Fig. 10.—Red-green test of Hess.

the green vertical numbers, giving the vertical deviation. He did not speak of using the test in the various positions of gaze, but it could well be used in that way.

Hess,[36] of Zurich, who in 1949 was awarded the Nobel Prize for his discoveries concerning certain functions of the brain, was as a young assistant one of the first [37]

36. 1949 Nobel Prize for Medicine, Medical News, J. A. M. A. **141**:789 (Nov. 12) 1949.

37. Hess, W. R.: Eine neue Untersuchungsmethode bei Doppelbildern, Arch. Augenh. **62**:233-238, 1908.

to popularize the use of the red-green test for the measurement of diplopia. He believed that the Hirschberg technique, which employed a candle and a coordinate system painted on a wall, was not practical enough for clinical use. He utilized a tangent screen made up of red lines on a black cloth [38] with red spots at intersections of the 15- and 30-degree lines with the red lines along the horizontal and vertical axes (Fig. 10). The patient held in his hand a wand with a green test object at its end; this green test object was later formed in the shape of an arrow. The head was held absolutely quiet, and only the eyes followed the object. A pair of red-and-green goggles was placed before the eyes. Thus, one eye, looking through the red filter, could see only the red spots and the lines on the chart; the other eye, wearing the green filter, could only see the green test object on the end of his stick. It was now the patient's task to indicate with his pointer the position of each red mark on the tangent screen. Since he saw the red mark with one eye and had to project the green arrow with the other eye, true superimposition of the green arrow on the red dot would occur only if there was no deviation. If a deviation was present, the separation could easily be demonstrated and measured.

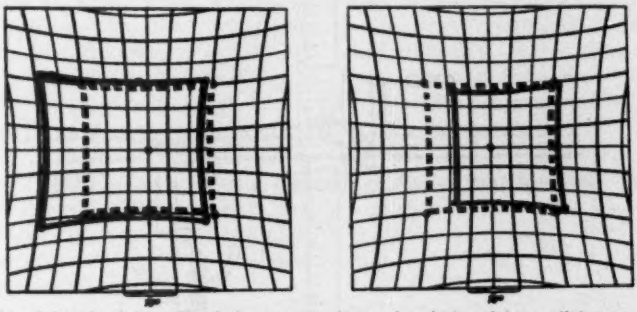

Fig. 11.—Diplopia fields (Hess) in a case of paresis of the right medial rectus. Left: Secondary deviation: right eye fixing (red); left eye (green). Note that the paretic right eye has the smaller field (red). Right: Primary deviation: left eye fixing (red); right eye (green). Note that the paretic (right) eye has the smaller field (green). The field for red is indicated by the broken line; the field for green, by the solid line.

Hess then plotted the relative position of the green arrow on the screen onto a similarly reproduced recording card (Fig. 11). Thus the test was made for the position of the nine red spots. The positions of all the red targets were then connected, and, in a similar manner, the positions of all the green targets were connected, forming two quadrilateral figures. Now, if the red-green goggles were reversed, the opposite eye would take up fixation, and the diplopia fields would then indicate the secondary deviation (if the first had measured the primary deviation). In each instance, the size of the figure for the paretic eye would be smaller than that of the nonparetic eye. Thus, one could differentiate a concomitant and a paralytic type of strabismus. Hess worked at a distance of 0.5 m., and he was careful in the construction of his tangent screen to make sure that he was working with true tangents; therefore, the lines appear curved and convex toward the center.

38. Hess, W. R.: Ein einfaches messendes Verfahren zur Motilitätsprüfung der Augen, Ztschr. Augenh. **35**:201-219, 1916.

Sattler [39] criticized the method of Hess as being deficient in that it made no measure of the torsion component; he also stated that too small a field was tested, and therefore one was likely to miss a weak paresis. He also objected to the distance of 0.5 m. because he felt that accommodation might play too important a role. He also criticized the charts that Hess suggested for recording as inadequate because the names of the muscles were printed on these cards, and therefore confused and made too small the area for recording. Ohm [40] felt that the 0.5-m. distance influenced the accommodation too much.

John Ohm, an assistant of Hirschberg, produced a number of tests for diplopia-field measurements. In one of his earliest works,[41] he utilized the principle of the complementary red-green filters by constructing a complicated apparatus made up of a vertical and a horizontal arm with movable green and red arrows. It measured

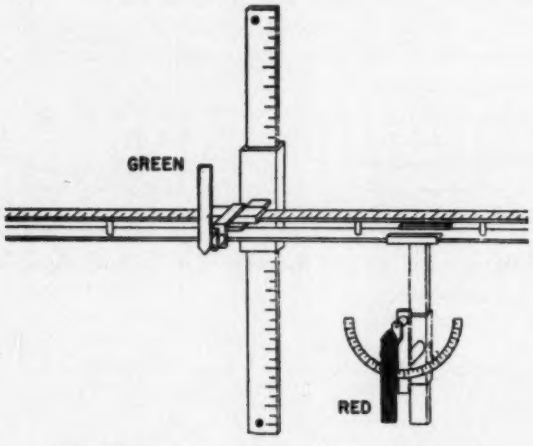

Fig. 12.—Apparatus for measuring diplopia (Ohm).

1.5 m. in height and 2 m. in width (45 degrees to each side of the zero point). The patient sat 1 m. away. Thus, by wearing the red-green glasses the patient saw one arrow with each eye. It was the task of the patient to indicate when the points of the two arrows touched and when the bodies of the arrows were in alignment. The red arrow was so mounted that it could be rotated according to the torsional displacement. With this, Ohm was able to measure the angle of deviation of the eye in the primary position and thereby indicate the extent of the diplopia. The primary and secondary deviations could be measured by causing fixation to be obtained with the nonparetic or the paretic eye (Fig. 12).

39. Sattler, C. H.: Über die genaue Messung und Darstellung von Bewegungsstörungen der Augen, Klin. Monatsbl. Augenh. **78**(pt. 1):161-179, 1927.

40. Ohm, J.: Das Ohrlabyrinth als Erzeuger des Schielens, Ztschr. Augenh. **36**:253-273, 1916.

41. Ohm, J.: Zur Untersuchung des Doppeltsehens, Centralbl. prakt. Augenh. **30**:326, 1906.

In 1907, he [42] referred to this apparatus as too clumsy and said that he was then substituting a method whereby he used a tangent screen after the method of Hirschberg [37] (1875), arranged in a coordinate manner. He worked at a 1-m. distance and used red and green paper arrows. The red arrow had its point down, and the green arrow had its point up; this device had the advantage that he could pin one paper onto any part of the field that he wished to test and then the patient would direct the position for the placement of the second paper seen by the other eye so that the two arrows pointed at each other. He utilized nine cardinal points where he would place the arrow.

Ohm [42] also devised a screen (1906) in which a black cloth was faced with blue strings spaced at 5-arc-degree intervals. The patient wore red-blue goggles and indicated the position at which the blue tip of a wand he held seemed to touch the part of a red arrow seen with the red-covered eye. Ohm [40] was impressed by the work of Krusius and liked the idea of the patient's being directly observed through the glass screen; in 1916 he reported a modification which permitted his apparatus to be used at a distance of 1 m. Krusius [43] tested at 0.5 m. Ohm stated that the accommodation at 0.5 m. influenced the test, and even influenced the labyrinth, and thus the distance was too short for his purpose, thus justifying his apparatus. He accomplished his aim by constructing a scaffold measuring 50 degrees to the right and to the left of the zero point and 25 degrees above and 45 degrees below the zero point. He strung this with wires separated at the estimated tangents of stated arc-degree intervals. Thus, he could stand on the farther side of the screen and, by looking between the wires, could observe the movements of the eyes and measure the angle of squint both objectively, by noting the corneal reflex, and subjectively, by utilizing the red glass and a white arrow in the various positions of gaze. The scaffold measured 250 cm. in width and 160 cm. in height and required the head to be fixed on a chin rest and the occiput fixed by a head rest.

Later, in 1921 and 1925, Ohm advocated the following method, which may represent what in his mind was the best of his tests from a practical and a clinical point of view. He utilized a chin rest and a head rest to keep the patient's head in a fixed position. The working distance was 1 m. He suspended [44] a white paper arrow, or made a small cross with white chalk at the zero point of the tangent screen, and placed a red glass before the deviating eye. The patient was then told to look at the point of the white arrow; this, of course, was done by the fixing eye, since the deviating eye was apparently sufficiently obscured by the red filter. He then held a small white illuminated bulb, which appeared red to the deviating eye. The bulb was so placed that the red light appeared to be on the point of the arrow; its position and distance from the arrow then gave immediately the angle of deviation of the squinting eye. The patient was told to ignore the white light, thus insuring bifoveal fixation. The arrow could then be used in different fields of gaze and the flashlight positioning repeated, the patient in each case disclosing the amount of diplopia by the distance between the light and the

42. Ohm, J.: Neue Instrumente, Medikamente usw.; ein Apparat zur Untersuchung des Doppeltsehens, Centralbl. prakt. Augenh. **31**:201-203, 1907.

43. Krusius, F.: Über ein Scheiben-Deviometer, Arch. Augenh. **59**:26-28, 1908.

44. Ohm, J.: Analyse des Doppeltsehens, Klin. Monatsbl. Augenh. **66**:20-39, 1921.

position at which it appeared at the point of the arrow or on the cross. Ohm [45] suggested that these findings be recorded on charts, the position of the deviating eye being indicated relative to the position of the fixing eye. After the manner of Hirschberg,[5] he marked the distance between these positions in degrees, designating a convergent position by a plus sign (+) and a divergent position by a minus sign (—); vertically, a right hypertropia was expressed as plus (+) and a left hypertropia as minus (—). By connecting the relative positions of these values on a graph by a line, he demonstrated his so-called strabismus curve (Fig. 14).

Sattler [39] used the complementary red-green test in the following manner: He employed a black wall, 2.4 m. square, on which was ruled a coordinate system painted with pure green lines. (This in itself offers great difficulty, since complementary green is not easy to produce.) The wall was ruled off very much like graph paper, in that the lines at the 10-degree intervals were a bit thinner than the two zero lines, and the lines indicating 5-degree separations were the thinnest. The zero point was placed 120 cm. from the floor, approximately at eye level. He

Fig. 13.—Wire-strung device for measuring diplopia (Ohm).

placed green marks (crosses) at the 20-degree crossings and at the zero point. The patient's head was secured by a chin rest and by an occipital rest, so that the head was vertical, erect and fixed, a point that he stressed as being most important for evaluating reexaminations. The patient held in his hand a long black stick at the end of which was a red cross, measuring 10 by 5 cm. Red-green glasses were placed before the eyes, and they were large enough not to restrict the field of vision. The spectacle frame was so arranged that it could be reversed for either eye, thus allowing a measure of primary and secondary deviations. The examining physician held a green stick in his hand, with which he directed the patient's attention for proper fixation at the target he wished him to look at (Fig. 15). The patient indicated this position with the red cross at the end of his stick. Since the cross was made up of vertical and horizontal components, the patient would unknowingly demonstrate torsion if such existed. The examiner

45. Ohm, J.: Blickfeldschema und Schielwinkeltabelle, Klin. Monatsbl. Augenh. **74**:311-324, 1925.

recorded immediately the findings on ordinary millimeter graph paper. Sattler then connected with a red line the points seen by the eye looking through the red filter and with a green line the points seen by the eye looking through the green filter (Fig. 16). He suggested that with a weak paresis one must test beyond a

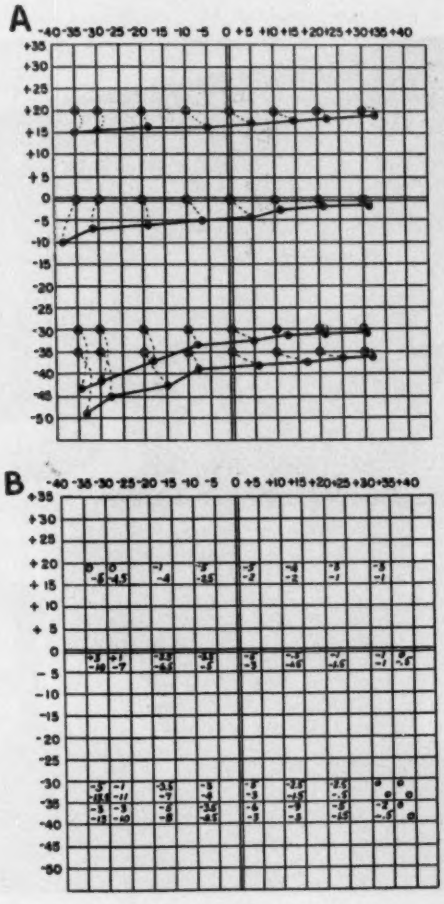

Fig. 14.—Strabismus curves (Ohm): *A*, paresis of left inferior rectus; *B*, paresis of left inferior rectus. In each case the left eye is fixing, and the red glass is before the right eye (secondary deviation).

20-degree field, but that with a more pronounced paresis a smaller field was sufficient. He made note that not much intelligence was required of the patient for an accurate result. It appears obvious that such a large screen immediately offers a difficult problem for the ophthalmologist in the usual office setup.

In 1939 Lancaster [46] described his red-green test. This was actually a streamlined application of the well-established principle employed in the previously described tests, using complementary colored targets and goggles. It would seem a practical apparatus, in which the best features of each were made available (Figs. 17 and 18).

Fig. 15.—Sattler's red-green test.

Lancaster used a white tangent screen, which could be rolled up out of the way. It was ruled into squares of 7 cm., so that at a distance of 2 m. each square subtended approximately 2 arc degrees, or 4△. The squares are all exactly the same size and extend 16 arc degrees to each side of the midline. If one wishes a

46. Lancaster, W. B.: Detecting, Measuring, Plotting and Interpreting Ocular Deviations, Tr. Sect. Ophth. A. M. A., pp. 78-94, 1939.

greater area, one may get this effect by bringing the patient to 1 m. from the screen, in which case the arc-degree values would be doubled. Lancaster made no allowance for the fact that the tangents would require the squares to be wider as one moved away from the center. He maintained that at a 2-m. distance rectangular squares are sufficiently accurate. The miniature reproduction of this screen on a card was used for the recording. The technique suggested is as follows: The patient holds a flashlight which projects a red linear light; he wears red-green

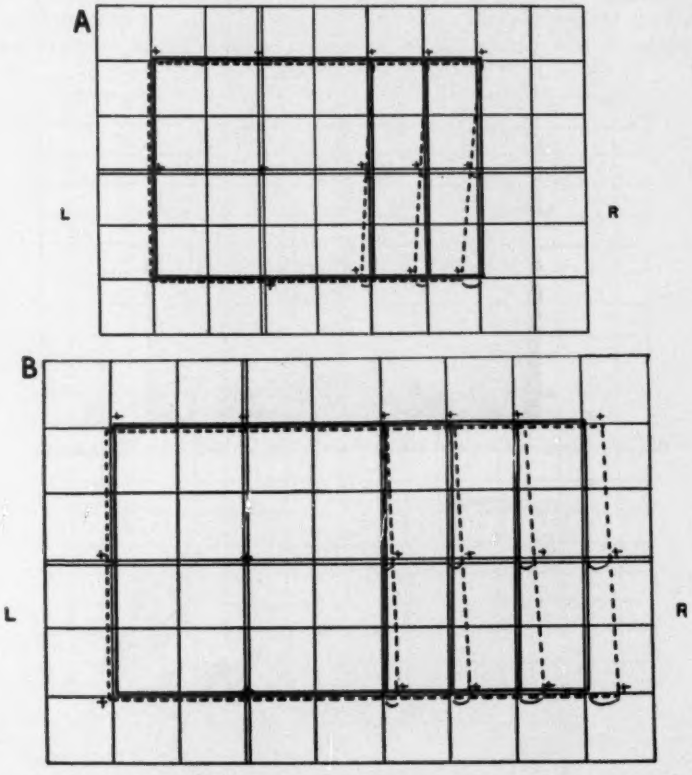

Fig. 16.—Paresis of the right lateral rectus (Sattler): *A*, left eye (green) fixing; primary deviation. *B*, right eye (green) fixing; secondary deviation. The field for red is indicated by the broken line; the field for green, by the solid line.

goggles, the red glass before the right eye. The examiner projects a green linear light in the various positions of gaze; the patient covers this projected green light with his red light. He sees the examiner's light with his left eye and projects his own light with his right eye. The readings are made direct; the displacement between the two lights is recorded on the chart. One notes that the lagging eye will actually lag; the divergence of the eye will give an uncrossed position of the two lights; the subject will project exactly in the direction of the eye. Lancaster

states that if one wishes a more extreme position of gaze than the chart will allow, the head may be turned in the appropriate direction. Thus, a patient wears red and green filters before the right and left eyes, respectively; his head is fixed, so that all movements must be from his eyes. The examiner throws one colored linear light onto the screen, and the patient covers that light with a similar, but

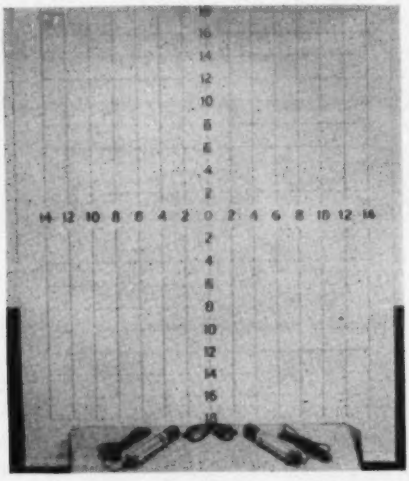

Fig. 17.—Equipment for Lancaster red-green test (Welch Allyn, New York).

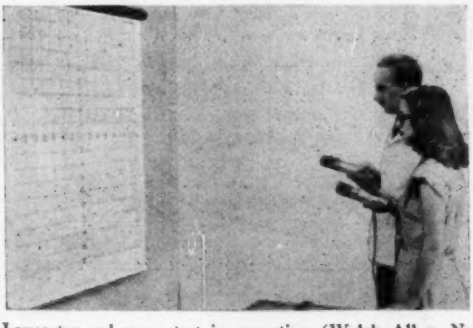

Fig. 18.—Lancaster red-green test in operation (Welch Allyn, New York).

complementary-colored, target. Seeing the examiner's light with only one eye and projecting the second light with the other eye, he is indicating the degree of his deviation. This test has strong advantages: first, it allows the examiner great liberty in finding the field of fixation that he wishes to test; second, by the use of the linear target, the effect of torsion is clearly and immediately indicated; third, it allows a measure of the secondary angle of deviation by exchange of the lights.

Lancaster deserves great credit for reducing the study of ocular motility in America to a level capable of popular reception and acceptance. His many papers [47] on this subject attest to the truth of this statement. His greatest work has been that of continually and repeatedly stressing basic facts in this field.

Anderson [48] modified the Lancaster red-green test in that he projected a chart, rather than using a curtain screen. His chart is plotted in squares of 7 cm., each subtending an angle of 2 degrees at 2 m. A red horizontal line is projected onto the center of the chart, and the patient is asked to cover this line with a green line that he projects from a torch he holds. Of course, he wears red-green goggles, so that each eye sees only one image. After the reading is made, the entire chart is

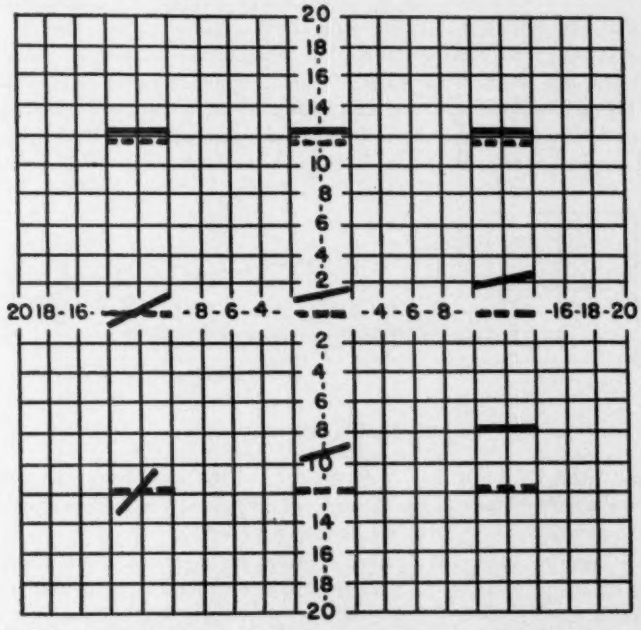

Fig. 19.—Method of recording in the Lancaster red-green test. Red glass before right eye (broken line); green glass before left eye (solid line). Note direct representation of extorsion in the left eye down and to left.

projected to another of the various positions of gaze to be tested, and the previous procedure is repeated. Thus, the red image is always at the center of the scale, providing for easy measurement. The results are plotted on ordinary graph paper.

Burian,[49] in a memorable paper on fusional movements in strabismus, described a modified diplopia test using polaroids® to produce monocularly seen targets. He

47. Regan, J. J.: W. B. Lancaster, M.D., Biographical Sketch and Bibliography 1897-1943, Am. J. Ophth. **26**:445-453, 1943.

48. Anderson, J. R.: Ocular Vertical Deviations, Brit. J. Ophth., Supp. 12, 1947, p. 80.

49. Burian, H. M.: Fusional Movements in Permanent Strabismus: Study of the Role of the Central and Peripheral Retinal Regions in the Act of Binocular Vision in Squint, Arch. Ophth. **26**:626-652 (Oct.) 1941.

employed two projection instruments provided with polarizing plates, one polarizing the light in the vertical direction and the other in a horizontal direction (Fig. 20). One instrument projects a tangent scale properly calibrated in arc degrees for 4.5 m., or 15 ft. The other instrument projects a colored dot; this projector is movable, so that the dot can be placed anywhere on the screen desired. The patient wears before each eye a square polarizing plate, each so oriented that one eye sees the dot and the other the tangent scale. The frame carrying these polaroid® plates can be reversed, so that either polarizing agent can be placed before either eye: Thus, one can change fixation to the opposite eye. At the beginning of the examination the dot is placed on the screen so that it coincides with zero of the scale. If both eyes are not directed toward the same point, one image will fall on an extrafoveal position, and a spot will appear on the scale at the angle of squint; thus, it may be measured directly. By moving the head so that the various positions of gaze are obtained, one may measure the angle of squint, or measurement of diplopia, in all fields. This may be done either by the patient's noting the apparent position of the dot in relation to the scale or by moving the dot so that it appeared to be superimposed on the center of the scale; in either case, the examiner reads the amount

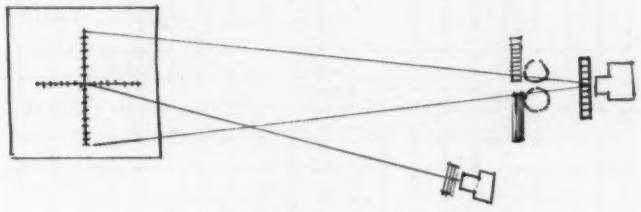

Fig. 20.—Diplopia-field test (Burian).

of diplopia directly. The amount of equipment and the setup required for this test would seem to make it one that might prove not very popular with most practitioners, who would have little or no other use for this apparatus.

Lees [50] recently described a rather complicated apparatus for measuring the binocular relationship. It employs the principle of bifoveal imaging and is said to be superior to the Hess test in that it does not depend on a color-separation system. Lees stated that if the colors "are not correct, the dissociation obtained is only of relative degree and errors then are liable to occur in the test." In his instrument, dissociation is obtained by a plane mirror placed before one eye. It has two reflecting surfaces, so that it may be used before either eye. The mirror is exactly halfway between two identically printed Hess tangent scales, erected at a 90-degree angle to each other. Each scale is so constructed that its pattern may be made to appear or disappear as required during the test. Thus, the patient looking at one scale directly and the other in the mirror sees "different but identical scales exactly superimposed." The eye being plotted has its scale disappear, leaving only a blank surface, in order to avoid fusional stimuli (Fig. 21).

50. Lees, V. T.: A New Method of Applying the Screen Test for Inter-Ocular Muscle Balance, Brit. J. Ophth. **33**:54-59, 1949.

The patient sits close to the mirror facing directly one screen 50 cm. away, while the other eye sees the second screen in the mirror. The mirror itself allows each eye to see only one screen. The examiner places his pointer at the various positions of gaze on the screen visible in the mirror. The patient, in turn, places a second

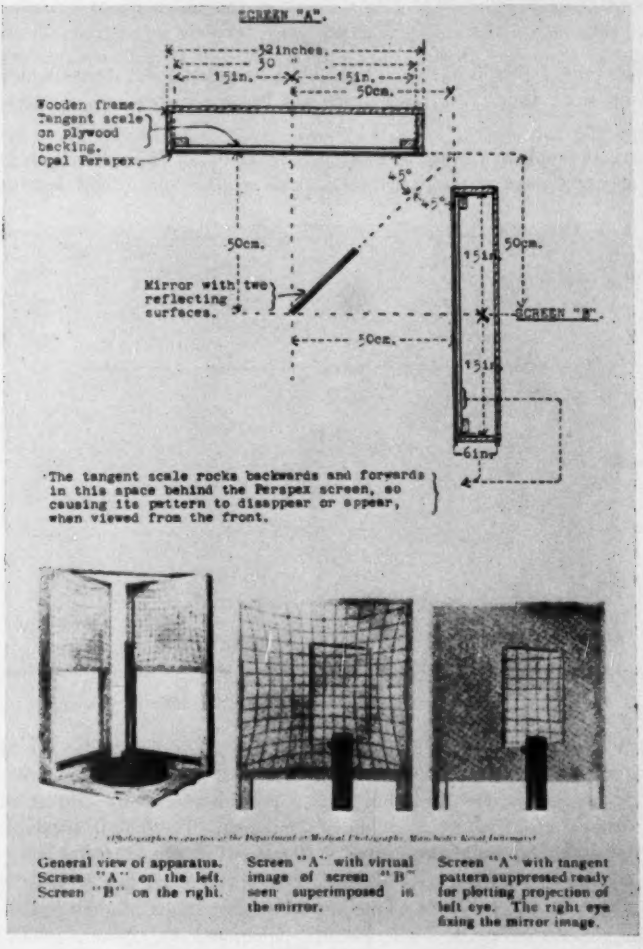
SCREEN "A".
32inches.
30 "
15in.
15in.
50cm.
Wooden frame.
Tangent scale on plywood backing.
Opal Perspex.
45°
50cm.
50cm.
15in.
Mirror with two reflecting surfaces.
SCREEN "B".
50cm.
15in.
61in.
The tangent scale rocks backwards and forwards in this space behind the Perspex screen, so causing its pattern to disappear or appear, when viewed from the front.
General view of apparatus. Screen "A" on the left. Screen "B" on the right.
Screen "A" with virtual image of screen "B" seen superimposed in the mirror.
Screen "A" with tangent pattern suppressed ready for plotting projection of left eye. The right eye fixing the mirror image.

Fig. 21.—Plan of mirror-screen test (Lees).

pointer where it appears to cover the examiner's pointer. Of course, he indicates these positions on the second screen, from which the scale has been made to disappear. By making the scale appear after each position is noted, a reading can be made directly. The findings are transferred onto the Hess chart for recording, as

with the red-green test. It is of paramount importance that the apparatus be accurately aligned. Lees stated:

> . . . It will be realized that, as the apparatus requires to be installed as a semi-permanent fixture, and as its manufacture is more exacting, the initial cost will far exceed that of the original Hess screen test. The advantages offered in rapid, accurate and trouble free results far outweigh the initial difficulties and expense involved.

NEW DEVICE

At this time, I should like to introduce for consideration a device which I have been using since June, 1949, and which has become increasingly valuable to me with daily use.

It consists simply of a board measuring 18 by 12 in., or 30 by 45 cm., and is made of a transparent piece of plastic material. It is mounted on a light-weight handle,

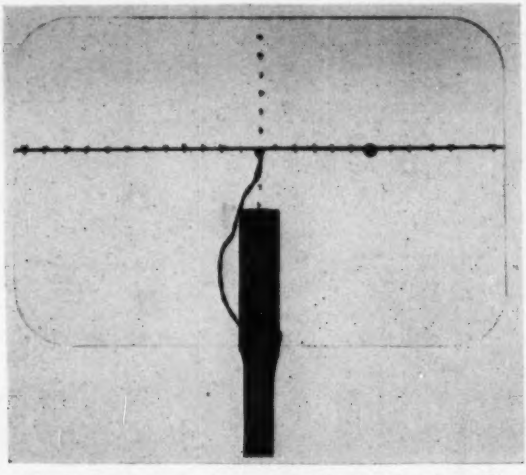

Fig. 22.—Device for measuring certain binocular functions (Sloane).

contains a small central bulb and has a Maddox cross, indicating the tangents up to 24 arc degrees to either side of the horizontal zero, and 12 degrees above and 16 degrees below the horizontal zero. The instrument is held at the easy working distance of 0.5 m., or 20 in., from the patient. The bulb is of the type normally found in a May ophthalmoscope; it is encased in a condensing lens, so that a bright corneal light reflex is obtainable. I have added a movable target so that certain other tests can be made. Thus, in effect, one has a Maddox cross, calculated in degrees for 0.5 m. on a transparent medium and containing a special source of illumination in its center (Fig. 22).

For measuring the diplopia, the patient's head is fixed securely against a head rest, although this is not absolutely necessary. A dark-red glass is kept before one eye; the patient holds an unsharpened lead pencil, using its rubber-eraser end as a pointer. The device is then kept 0.5 m. from him, the observer standing directly

behind, so that he may watch the movements of the eye (Fig. 23). The patient then indicates the position of the red light by placing the eraser at the end of the pencil onto the device: Its position is easily noted and plotted, the degrees of diplopia being indicated directly, both vertically and horizontally. The patient's head is then turned in the various positions of gaze. It is quite easy after a short period of practice to determine how far the patient's eyes can be turned and yet not have obstruction from the nose or facial configuration. This is possible because the observer stands directly behind his light and notes the presence of the light reflex on the cornea of both eyes. When the eye is turned too far to one side, so that, for example, the nose obstructs, he does not see the light reflection on this eye; therefore, the head of the patient is rotated until the light just appears. The working distance is convenient; the patient indicates easily, and surely, the position of the red light. The measurements of diplopia are made in the various positions

Fig. 23.—Application of diplopia test (Sloane).

of gaze, and the test is accomplished in very short order. In the matter of diagnosis of an ocular paresis, I have found no disadvantage in using the test at a 0.5-m. distance as compared with the 2-m. distance that I had been using with the double-image type of test for the past 11 yr.

In a patient who is unable to move his head, as may occasionally be the case when one is examining an arthritic, the device may be moved in the various positions of gaze. Its portability makes it convenient to use elsewhere than in the office. I have found it particularly helpful in examining patients in consultation in the hospital.

After having used this device for some months, I found a similar one described by Krusius [43] in 1908. His *Scheiben-Deviometer* consisted of a pane of glass 120 by 60 cm., or 48 by 24 in., upon which was calibrated a cross computed for tangents in arc degrees. The screen was further divided into small squares. A working

distance of 60 cm., or 24 in., was employed. He used two luminous bulbs for fixation objects and watched the position of the corneal reflex in the determination of the angle of strabismus (for which this instrument was intended). The alignment of the corneal image was executed by flashing off and on the bulbs, one of which was movable and could be centered with the angle of squint. He said an allowance of 6 degrees should be made for the convergence at 60 cm. Its size and weight and the fragility of the glass probably explain why the device is not portable. On the basis of Krusius' work, Ohm [40] made his wired-tangent scale, previously described.

COVER TESTS

The cover test, utilizing so-called parallax, is a subjective test that may measure diplopia by use of prisms. In this technique, the patient will observe a movement in the object viewed when the cover is changed from one eye to the other. The amount of this movement, of course, is determined by the amount of excursion that the eye must make to recover its position of fixation. Prisms introduced appropriately will neutralize the apparent movement of the object when alternate occlusion takes place. The end point would then be determined by the patient's stating when there was no movement of the target as the two eyes were alternately covered. Duane [13] commented:

> . . . These movements are noticeable even when the deviation is very slight: a hyperphoria of 0.1 degree being made appreciable by a distinct up and down movement of the object.

Von Graefe [51] stresses that the prisms which will just neutralize the diplopia will vary, and that the prisms which will hold the vision single easily are the true measure. Landolt,[10] too, suggested prisms used in this way as a measure of diplopia, although most of his experiments [18] in this field dealt with the use of a tangent screen of tremendous size, employed at a distance of 3 m. White [52] described this cover test as dependent on the cooperation and on the observations of the patient.

> . . . Many patients are quite stupid about being able to interpret the movement of the test. . . . It is a very delicate test when properly observed, and again is quite tedious and valueless when cooperation is lacking.

He found that he "used it much less than formerly."

Maddox [52a] believed that his objective method of measuring the angle of squint combined the accuracy of the perimeter with the speed of the Priestley-Smith [53] method. He used a tangent cross calibrated for 1 m., in the center of which was a lighted candle. While the patient fixed the candle, he noted the position of the corneal image in the deviating eye, this giving opportunity to estimate quickly the angle of squint by the Hirschberg [54] method. The sound eye was then covered to

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51. von Graefe, A.: Motilitätsstörungen: Die Lähmungen der Augenmuskeln, in von Graefe, A., and Saemisch, T. T.: Handbuch der gesamten Augenheilkunde, Ed. 2, Leipzig, W. Engelmann, 1898, Vol. 8, p. 45.

52. White, J. W.: The Screen Test and Its Modifications, Screen Maddox Rod and Screen Comitance, Am. J. Ophth. **24**:156-160, 1941.

52a. Maddox, E. E.: Objective Strabismometry, Arch. Ophth. **21**:56-62, 1892.

53. Smith, P.: A Tape Measure for Strabismus, Ophth. Rev. **7**:349-352, 1888.

54. Wheeler, M. C.: Objective Strabismometry in Young Children, Arch Ophth. **29**:720-736 (May) 1943.

allow the squinting eye to fix the candle, and when it did this the position of the corneal image was noted. The dominant eye then was again uncovered, and the patient was told to look at the numbers suggested by the Hirschberg estimation; if this was corect, the corneal image was noted to be in the center of the deviating eye. If this was not the case, the dominant eye could fix on the proper number until the candle light was reflected in the center of the deviating eye. This figure gave the measure of the squint in its primary deviation. The paretic eye being caused to fix, the secondary deviation was measured in a similar manner. Maddox did not enlarge on the use of this technique for the different positions of gaze, but its application to such measurements is, of course, obvious.

With the device that I have described (Fig. 22), I can obtain a reasonable determination of the angle of squint without the use of prisms. First, the patient's head (as in the diplopia field) is kept in a stable, fixed position if possible. Then, looking directly from behind and sighting along the light of this device, standing 0.5 m. from the patient, I observe the position of the reflection of this light on the cornea of the deviating eye while I have the fixing eye occluded.

This method at once establishes several facts: 1. Angle kappa is not a factor, since I am determining my point of reference for the corneal reflex by the visual axis. Now, I have my light centered where it should be centered if the eye is working in a fixing position. This will be my point of reference, and here I shall try to place the same light when I make my measurement. 2. Since I am sighting along my light, I am essentially normal with it, so that rays of light reaching my eye from this corneal image practically retrace their path from the light, at least in the horizontal plane. In this way, I shall not be confused by any consideration for the angle of reflection, and no allowance need be made for that. Now, I uncover the fixing eye. Of course, this light reflection is now displaced (according to the angle of squint) away from the center. I then move a target, temporarily a colored button, across the face of this board, asking the patient to watch the button. This, of course, he does with his fixing eye. I move the button until I note that the light reflex is again in the center, or at that point of the cornea where I noted it originally in determining my point of reference on the deviating eye. The deviation will then read directly from the position of the button. If I wish to check this, I simply cover the fixing eye, which had been looking at the button, and direct the attention of the patient to the light. Since the deviating eye is now directed toward the light, no movement should be noted; if there is, I am not absolutely accurate, and I can make my adjustments by moving the target to one side or the other. This is no different from utilizing the technique as presented for any of the major amblyoscopes, whereby the target for each eye is set for the objective angle of squint, and one has arrived at the measure when one obtains no movement when the eyes are alternately covered. Now, to measure the angle of squint in the various positions of gaze, I turn the patient's head in the appropriate directions.

Krimsky [55] observed the displacement of the corneal light reflex from the fixation position in the deviating eye. He then measured this deviation by the amount of prism necessary to restore this light reflex to its fixation position. This test was then repeated in the various positions of gaze. Thereby the field of greatest disjunctivity of visual axes was found and evaluated.

55. Krimsky, E.: The Management of Binocular Imbalance, Philadelphia, Lea & Febiger, 1948, p. 206.

The cover test is associated with the name of Duane. Here, one looks for the field of greatest recovery required (when the screen is removed) as the field of action of the paretic muscle. One can measure with prisms the amount required for neutralization of this movement and thus obtain a quantitatively accurate reading. This test must be done in the six cardinal fields and is most important in the diagnosis of paresis, even of a slight amount. A convenient working distance suggested by White is between 15 and 18 in. (38 and 45 cm.). It is frequently found that the deviation, measured with prisms, is greater when the prism is placed before one eye than when placed before the other. This is due to the difference between the amounts of the primary and secondary deviations. An indication as to which is the paretic eye may be obtained by noting that a paretic eye is the slower-moving eye, whereas the faster-moving eye is so because of the greater secondary deviation. White [56] recommends that for a successful use of the cover test the following factors are requisites:

1. There must be central vision in each eye.

2. Sufficient light must be present so that the examiner may note the least movements of the eye, but the light must not be so bright or glaring that the patient or the observer is blinded by it.

3. The screen or cover should be large enough to cover an eye without undue attention to its position. White prefers a card 6 by 15 cm.

4. The cover should not be moved more rapidly than the uncovered eye can take up fixation. The square prism is most satisfactory, and the usual size, of 4 cm., is sufficient. He states that the round prisms are unsatisfactory, since they are hard to hold separately and impossible to hold in combination. He does not advocate the rotary prism. If cooperation is good and fixation normal, an amount of strabismus [56] of 80 to 100△ may be accurately measured.

Cover testing with prisms is simple in theory, but not always easy in practice, particularly for those persons who have not long used them and who may not have the dexterity required to hold a number of strong prisms in their proper position before the eye. This excellent method of measuring diplopia requires that the examiner have some practice with the use of prisms. It is with repeated use that the cumbersomeness and awkwardness of the various manipulations disappear. One must be able to use the cover before the alternate eye, at the same time making sure that the patient keeps his fixation in the proper direction. The examiner must also be deft in the handling of prisms, so that in a minimal amount of time the prisms may be put into the right position. The error produced if the prisms are not held in their proper position is not always easy to avoid. The cumbersomeness of these manipulations in the hands of the occasional user will, unfortunately, sometimes make the ophthalmologist skip this excellent test. No doubt, the prism bar, as described by Berens,[57] is doing much to oppose this tendency. Great credit must be given to Dr. White's influence in making this method a popular one in this country.

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56. White, J. W.: Clinical Application of the Screen (Cover) Test Described in Detail, Am. J. Ophth. **27**:977-986, 1944.

57. Berens, C.: A New Prism Bar, Am. J. Ophth. **22**:305-306, 1939.

In regard to this cover test, Ludvigh [58] points out certain limitations of ability to note small movements of redress:

> . . . Under ideal conditions, excursions of much less than 1 D to 2 D cannot reliably be perceived by the unaided eye. In usual clinic conditions and with much less coöperation from the examinee, it seems advisable to adopt at least the upper limit of 2 D. This means that if it is desired to detect eye movements of 1° or less, either a subjective test should be employed or the examiner use magnification.

CONCLUSIONS

After reading the literature and familiarizing oneself with the various techniques, one cannot help but formulate some conclusions as to the relative merits of the various diplopia-field tests. The following statements represent my impressions at this time.

1. Any person who has a working knowledge of ocular motility and the principles of projection of images will have no difficulty in analyzing and evaluating the diplopia fields by any of the techniques.

2. A personal interest in a given test will make that test particularly desirable and allow the person to put up with the objectionable features and enlarge upon the favorable aspects.

3. In analysis of the subjective methods the following factors are significant:

A. The testing distance. In the literature studied, this has varied from 0.5 m. (20 in.) to 6 m. (20 ft.). The former has been criticized as being conducive to the stimulation of accommodation,[39] with its influence on the angle of deviation and convergence.[42] The latter is too great to permit one to observe the eye if one is using a movable light with the head in a fixed position, and the scale is inadequate in degrees of extent if one uses a method such as the Maddox cross. A mean average for 15 methods studied was 1.8 m., or 72 in. Thus, on the basis of desirable distance arrived at by such means, one might choose the Bielschowsky technique, of the tests employing double images (unifoveal and extrafoveal imaging) and 2.5-m. distance, and the Lancaster red-green test, of the complementary color tests (bifoveal imaging) at 2 m. The Anderson modification of Lancaster's test would also apply.

B. Type of scale. All but one of the methods studied used tangents of arc degrees, this providing larger intervals for equal arc degrees as one went away from the zero point. Lancaster, however, insists that for practical purposes at 2 m. rectangular squares [46] are sufficiently accurate. It appears to me that from a clinical viewpoint the result would not differ appreciably one way or the other. Since we think in terms of exactness, even though we know that we cannot measure to this degree, it might have been better for the Lancaster chart to have been ruled into tangent equivalents. This, however, is an insignificant criticism in the over-all picture of an excellent test.

C. Moving the head, or moving the test object. Of 16 methods, 11 showed preference for obtaining the eye movements by moving the target. Four chose a fixed test object and moved the head. Lancaster moved the target but stated that if one wanted a wider excursion than this chart offered, the head [46] could also be

58. Ludvigh, E.: Amount of Eye Movement Objectively Perceptible to the Unaided Eye, Am. J. Ophth. **32**:649-650, 1949.

turned, but turned a precise amount, namely, to another fixation on the chart, which becomes the new zero point. Bielschowsky was a powerful force in advocating the head movement, because in this way he could control the situation himself. Actually, head turning does not provide for as repeatable a measurement as moving the target to definite positions. The possible objection of labyrinth stimulation in head movements has been raised, but appears to be unfounded by actual practice. I use head movements most of the time, but may feel prejudiced in its favor by Bielschowsky's influence and my personal interest in this test.

D. Unifoveal and extrafoveal imaging versus bifoveal imaging. The complementary red-green tests have found many disciples, as witness the large number of papes written, and now have had a rebirth in this country by way of the Lancaster version. The red-glass double-image test associated with a Maddox scale has many followers, too, largely through the influence of Bielschowsky and his school. It would seem that one weakness in the red-green test is, paradoxically, one of its principal claims to superiority. As has been stated many times, the positioning of the images in the complementary red-green test is direct; i. e., esotropia produces crossed images, and exotropia, uncrossed images. It is true that such a concept is much easier to teach the neophyte, who often finds it difficult to grasp the apparent reversed positioning of the images. On the other hand, the diplopia with the red-glass test is in keeping with the experience of the patient who has double vision: A person with paralysis of the sixth nerve does have homonymous diplopia, even though his visual axes cross. This source of confusion is readily eliminated if one considers the deviation only, and does not think in terms of diplopia. Actually, in the red-green test there is no subjective diplopia, the patient having superimposed one image on the other. Nevertheless, one must be on guard, for if one carries out tests for other functions utilizing double images, e. g., the Maddox-rod, the von Graefe's near-phoria (prism) test, or the square-prism test, the reverse positioning must be interpreted properly (unless one decides to substitute all these tests for those employing the complementary red-green principle).

E. Size of the scale. In the literature reviewed, the scales have varied from one of the size of an entire wall to a plastic board 18 by 24 in. (45 by 60 cm.). The longer the test distance, the greater the area of the scale required. Actually, normal eye movements are those never exceeding a 12-degree [59] excursion; but in testing for a paresis, one goes well beyond these limits, in order to limit a given field as much as possible to the action of a single muscle. Now, if one prefers moving the head, a relatively small chart is required, whereas if one favors moving the object of fixation, a much larger chart is necessary. For example, the coordinate type of screen used by Sattler and Ohm embraces amplitudes of as much as 50 degrees to either side of the zero point. From the standpoint of mere practicability, so far as the spatial limits which the usual examining room permits, use of head movements is a desirable method for getting the most ocular excursions for the size of chart used.

F. The cover-test neutralization of apparent movement of the test object by prisms. This was one of the earliest tests described; certainly, enough years have passed to have given it its proper place. White,[52] who had had a greater experi-

59. Fischer, F. P.: Über die Verwendung von Kopfbewegungen beim Umhersehen, von Graefes Arch. Ophth. **113**:394-416, 1924.

ence with cover tests than most people, put it reasonably when he stated: "It is a very delicate test when properly observed and again is quite tedious and valueless when cooperation is lacking." He found that he "used it much less than formerly."

G. Dissociation of the images by polaroids®: Essentially, this type of test has been used in the laboratory as part of the investigation of other phases of binocular functions. Thus, a true evaluation of it in this field has not been available. However, there is no reason to believe it would not serve its function well. One must consider the expense of providing the projection apparatus and equipment required, although this cost might well be brought to acceptable limits if the apparatus were produced in quantity.

H. Overlapping of double images and measurement of separation of the two test objects. Not enough experience has been had with this technique, as suggested by Kestenbaum. One would imagine that the influence of torsion might offer some confusion, since dissimilar test objects are employed. From the standpoint of a qualitative, rather than a quantitative, method, and one that requires no special equipment, it has its specific merits.

I. Shape of the fixation object. The issue resolves itself into an arrangement whereby, in addition to the separation of the images, the influence of torsion may be demonstrated, or by which the separation of the double images alone will offer the necessary diagnostic clues. The linear test objects, such as Sattler's cross, Ohm's arrow, and Lancaster's line, all satisfy the first situation. The round source of light only satisfies the second situation. Of course, if one uses the spot of light for the diplopia and, when indicated, a horizontal line for the torsion, as suggested by Bielschowsky, both conditions are satisfied, but two tests are required, as against one when a linear test object is used. The reason that the spot of light has been able to survive as a practical test object is probably due to the fact that in the evaluation [60] of a paretic muscle diplopia is of prime importance and the torsion of secondary importance. (It is not my intention to underestimate the great importance of torsional disturbances.)

Comment.—It seems to me that there is a place for a test which can be made at an easy-working distance, such as 0.5 m. The device that I have described seems to offer a considerable amount of information when used at the 0.5 m. distance. Frequently, the practicability and the convenience of this test may be of sufficient advantage to offset the disadvantages of the exercise of some convergence and accommodation. This is not to imply that other tests for distance should be omitted in favor of such a test; rather, one may well augment the other in everyday practice.

4. The objective methods employed the following principle:

A. Utilization of the corneal reflex in a centered position as the measure of the angle of deviation. The Krusius apparatus, with its switch-controlled two lights, apparently was not used enough, if at all, in the different positions of gaze. Its size, weight, and fragility (glass) must have handicapped its use in this test. Ohm's wire-strung coordinate system was so large that it probably would not

60. Mauthner, L.: Die Lehre von den Augenmuskellähmungen, Wiesbaden, J. F. Bergmann, 1899, p. 527.

appeal to the clinician, provided another, equally good method was available. Also the 1-m. distance between examiner and patient would make it difficult for the examiner really to appreciate the position of the corneal reflex. The Krimsky technique of cover and prisms for centering the reflex is good within the limits of any corneal-reflex technique. It has the advantage of allowing both distance and near fixation. The test I have described also lends itself to measurement of the deviation by observing the centered corneal reflex of the light, but is limited to near distance. The corneal-reflex method may not be quite as accuratae in reaching a decisive end point, but it is rapid and easy to perform.

B. Neutralization with prisms of the movement of redress when the eyes are alternately covered. This test appears to be the most accurate of the objective tests, since in the alternate occlusion each eye must take up fixation, and thus neutralization of the movement represents the measure of the deviation separating the fixations of each eye. If it were possible to reduce the intrinsic awkwardness of the handling of a large number of strong prisms in their poper positions, a great service would be rendered ophthalmologists.

5. Methods of recording data are not uniform. Practically all authors until the period of Peter [61] plotted their charts so that the patient's right was also right on the chart. White, like Peter, also plotted his chart as he looked at the patient; thus, the patient's right is on the left side of the chart. Advocates of the red-green test plot the right eye on the right side of the chart. Official agreement might tend to lessen confusion. (A similar situation previously existed in plotting visual fields and the axis of astigmatism.)

From the above discussion, it would appear that, of the subjective tests, the red-green test as presented by Lancaster and the red glass used and advocated by Bielschowsky more nearly represent the total best points of all the tests reviewed, and that the cover test for neutralization of movement of redress with prisms, as advocated by Duane and stressed by the White school, is the best accepted of the objective tests. Duke-Elder [62] states:

> . . . A measurement of the primary and secondary deviation can be obtained by neutralization with prisms, more easily and accurately by the double images of the tangent scale and most easily by the Lancaster projection test.

I cannot but think that in average hands the subjective tests are the most effective.

The conclusion is inescapable that more than one method is necessary to the person who would be well equipped, and that familiarity with the principles of the various tests can only make for a better understanding.

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61. Peter, L.: Extra-Ocular Muscles, Philadelphia, Lea & Febiger, 1941, p. 248.
62. Duke-Elder,[32] p. 4040.

ANALYSIS OF OCULAR MOVEMENTS BY MEANS OF AN ELECTRICAL METHOD

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THE PRESENT investigation is directed toward a study of the eye movements and the extrinsic ocular muscles during movements. Such movements have been studied extensively and have recently been reviewed by Marg.[1] The methods used consisted substantially in the recording of the electrical potentials, picked up by means of electrodes placed behind the external canthi of the subject, showing the movement of the eyes. Fenn and Hursch[2] have demonstrated that a relationship exists between these potentials and the angular position of the eyeball. These methods do not permit a simple analysis of the velocity and acceleration of the eyeball at any time. The method communicated in the present paper is intended to give a direct record of these physical magnitudes, and it also permits measurement of the forces exercised by the extrinsic ocular muscles.

In these experiments the cornea-retinal potentials are picked up with two electrodes, amplified, and recorded (displacement, first horizontal row of Figure 1). However, in addition, the amplified signals are electronically differentiated (second horizontal row). The resulting record furnishes a graph indicating the rate of change of the eye position, i. e., the velocity of the eyeball. A second differentiation (third horizontal row) furnishes the rate of change of the velocity, i. e., the acceleration of the eyeball. Since the force moving the eyeball is proportional to the acceleration, the last record is a measure of the resultant force exercised by the extrinsic ocular muscles. The method is described in detail by Powsner and Lion.[3] The two sets of schematic graphs shown in the vertical columns *A* and *B* may further illustrate the method. The fast change in the eyeball position in column *A* is reflected in the large deflection in the velocity record, and the high acceleration (+) and deceleration (—) of the eyeball. Column *B* shows the same records for a (hypothetical) slow motion. The advantage of the method consists in the complete presentation of the physical behavior of the eye during motion.

Experiments were made on 11 normal adults, ranging in age from 18 to 32. The subjects were all within normal limits with regard to correctible vision, binocular muscle balance, fusion faculty, convergence and gross extraocular movements, and ophthalmoscopic findings.

A typical reading pattern obtained is shown in Figure 2. The displacement tracing shows the return sweep (arrow), followed by five fixation movements

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1. Marg, E.: Development of Electro-Oculography: Standing Potential of the Eye in Registration of Eye Movement, Arch. Ophth. **45**:169-185 (Feb.) 1951.

2. Fenn, W. O., and Hursch, J. B.: Movements of the Eyes When the Lids Are Closed, Am. J. Physiol. **118**:8, 1937.

3. (*a*) Powsner, E. R., and Lion, K. S.: Testing Eye Muscles, Electronics **23**:96 (March) 1950; (*b*) A New Ergographic Method for Testing the Ocular Muscles, J. Applied Psychol. to be published.

(saccades). The velocity in the second tracing (*C*) is about three times as great for the return sweep as for the saccadic movements, as is also the acceleration, seen in the third tracing (*D*).

All records show a brief reverse movement, which constitutes the initial part of each voluntary eye movement. The reverse movement is particularly evident in Figure 3, which shows a movement of the eye from the center (primary position) through 38 degrees to the left, then back to the center, and through an angle of 38 degrees to the right. The arrow (*X*) in Figure 3 points to the small reverse move-

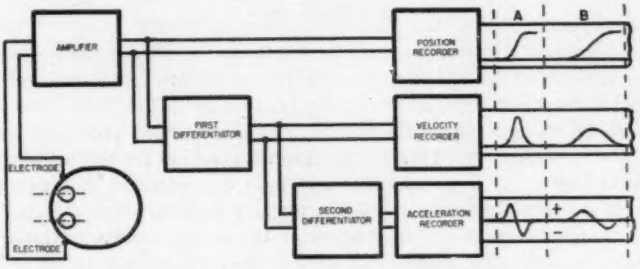

Fig. 1.—Schematic diagram of the experimental setup for the investigation of eye movements. For explanation, see text.

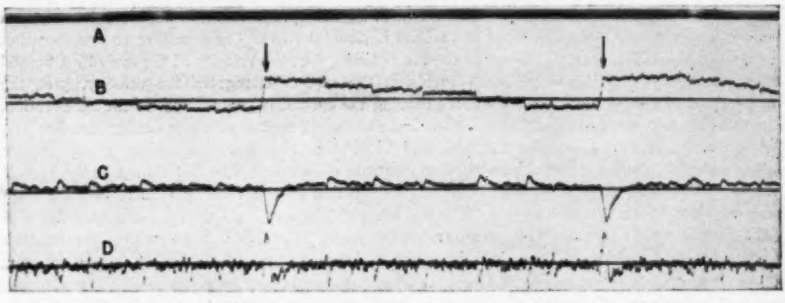

Fig. 2.—Reading pattern, showing a timing signal (*A*), the record of displacement (*B*), velocity record (*C*), and acceleration record (*D*).

ments to the left which precede the large deflection, representing a movement from left to right. Preceding the next gross movement, from right to left, the arrow (*Y*) points to the small reverse movement to the right. The presence of these reverse movements were observed before by Powsner and Lion [3b] and could be confirmed in all our records.

The record of Figure 3 is particularly noteworthy for the indication of greater velocity of the eye movement from a peripheral point to the center than for a movement from the center to either side. While this effect is small for small angular displacements, it becomes rather marked for eye movements through larger angles. The velocities of individual voluntary eye movements through different angles are

plotted in Figure 4. The greater velocity of movements toward the center than of movements toward the periphery is readily seen and is most marked at larger angular displacements. This effect is probably caused by the variation of torque arising through check ligaments and through the resistance of the relaxing muscle at large-angle deflections.

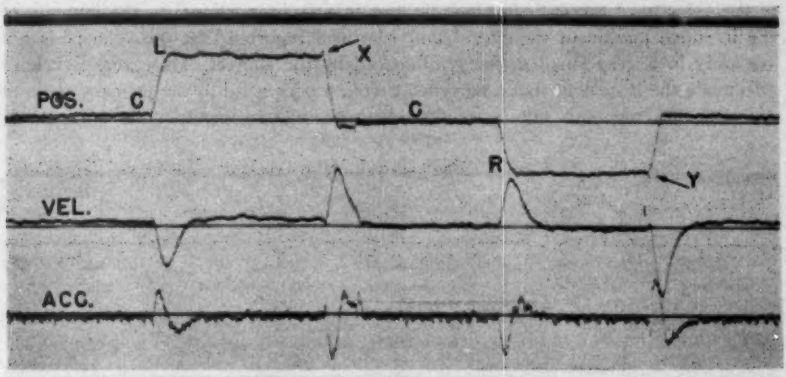

Fig. 3.—Record obtained by an eye movement of 38 degrees from the center to left, back to the center, 38 degrees to the right, and back to the center.

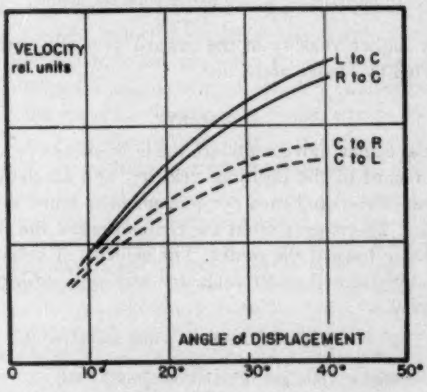

Fig. 4.—Eye velocity of a movement toward the periphery and that of a movement toward the center.

One of the particular characteristics observed throughout the experiments is the invariability of the velocity in any one subject moving his eyes between two marks through an angle of 90 degrees. Two alternately flashing lights were used as such marks, and the subject was asked to fixate these lights. The rate of flashing was varied from a very low frequency, of 1 cycle per three seconds, up to a point beyond which the subject could no longer follow (about 3 cps). The results (Fig. 5) show that the velocity remains constant, regardless of the rate of flashing, and that

only the time of fixation diminishes. The subject has no ability to change the velocity of the eye movement when voluntarily moving the eye from one point in space directly to another. When told to move the eye slowly from one point to another, the subject usually produced split movements. The velocity of each movement, however, was unaltered.

Our findings are in agreement with those of Brückner,[4] who found an increase in the velocity of the eyeball which is "not exactly proportional to the increase of the intended motion of the eye," [5] and who also reported that the velocity is not (or only in a very small degree) influenced by the subject. However, Brückner also made the statement that a movement from a peripheral to the primary position (inward movement) is not faster than an outward movement, while our records

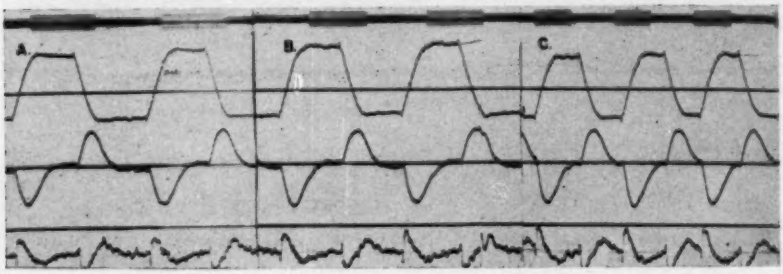

Fig. 5.—Records obtained by eye movements from left to right through an angle of 90 degrees; *A*, 105 movements; *B*, 128 movements; *C*, 170 movements, per minute.

show a remarkably higher velocity of the inward over the outward movement, as was formerly reported by Dodge and Cline.[6]

SUMMARY

Studies of normal ocular movements are made by means of an electrical method which furnishes a record of the position, velocity, and acceleration of the eyeball as a function of time. Preceding each eye movement is found a short movement in the reverse direction. Movements from the center toward the periphery are found to be slower than those toward the center. The velocity of voluntary movements is found to be characteristic and unalterable for any one subject and for any one angle of eye movement.

The Illuminating Engineering Society Research Fund sponsored this research.

4. Brückner, A.: Pflügers Arch. ges. Physiol. **90**:73-93, 1902.
5. Brückner,[4] p. 87.
6. Dodge, R., and Cline, W. W.: Psychol. Rev. **8**:145, 1901.

STUDY OF OCULAR MOVEMENTS UNDER STRESS

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AND
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IN THE preceding paper[1] a method was described for electronically recording angular displacement, velocity, and acceleration of the eye. This method makes it possible to study not only the eye movements themselves but also the forces that come into play during these movements. The present investigation concerns itself with the study of these movements, and the forces causing them, during stress.

Investigations concerning eye movements under conditions of stress and ocular fatigue have been made frequently in the past.[2] The result of much of this work is still unsatisfactory, especially in regard to fatigue. While some investigators find objective, as well as subjective, indications of fatigue, others have found no signs of impairment of characteristic ocular performances. It seemed interesting, therefore, to measure the work done by the extrinsic ocular muscles. The above-mentioned dynamic method which measures the acceleration of the eye is particularly suitable for this purpose, since the acceleration is proportional to the net muscular force causing the eye movement. Such a measurement might furnish a direct indication of the muscular impairment under stress. Failure to find such an impairment would indicate that fatigue, which is undoubtedly present, is to be found at another level of the complex process of vision.

The experiments were caried out in the following manner: The subject was seated, with chin in the conventional slit-lamp chin rest for head stabilization, and was directed to fixate either one of two small neon glow lamps, placed 45 degrees on either side of the horizontal plane. The glow lamps were alternately flashed, and the subject was to move his eyes from one lamp to the other as soon as he saw the flash of light. The rate of flashing was increased so that the subject was just able to keep up the pace, but also to move his eyes through the full 90 degrees of excursion. The rate of flashing varied from 120 to 152 movements for the different subjects. This kind of a test is much severer than a reading task, since during reading the eyes are moving only about 10% of the time; the remaining 90% of the time is consumed by the fixation pauses, during which the muscles relax.

Eleven adult subjects, of ages from 18 to 32, were examined. All were within normal limits in regard to correctible vision, ophthalmoscopic findings, binocular muscle balance, extraocular movement, and fusion faculty. They were told only that we wished to observe their eye movements.

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From the Laboratory of Applied Biophysics, Massachusetts Institute of Technology.

1. Brockhurst, R. J., and Lion, K. S.: Analysis of Ocular Movements by Means of an Electrical Method, A. M. A. Arch. Ophth., this issue.

2. Extensive references to the literature may be found in Carmichael, L., and Dearborn, W. F.: Reading and Visual Fatigue, Boston, Houghton Mifflin Company, 1947.

The test was preceded and followed by a short record of the reading pattern and a calibration of the eye movements through fixed angles, left and right.

The point of termination of the test was by necessity under the control of the subject. When he indicated that he was no longer able to keep up the pace or move his eyes successfully to the mark, the test was stopped. In some cases the subject complained also that ocular discomfort, burning, and watering prevented his continuing the test. In addition, some complained of nausea

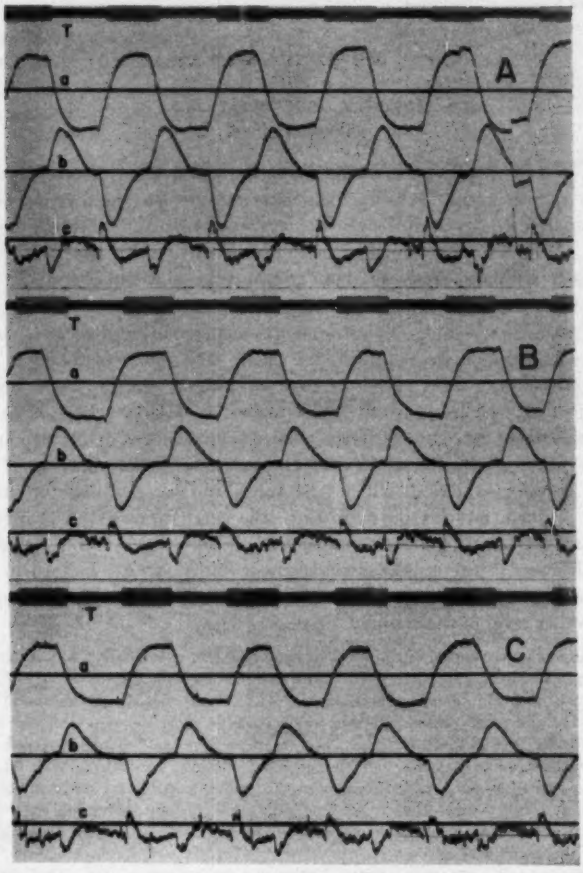

Fig. 1.—Record (Subject 8) of excursion (*a*), velocity (*b*), and acceleration (*c*) of the eyeball under stress: row *A*, performance at start of test; row *B*, performance after five minutes; row *C*, performance after 10 minutes. *T* is record of timing signal.

and dizziness. No true vertigo or nystagmus resulted. The stress period varied from four to 28 min., with an average of 10 min.

Figures 1 and 2 show typical records obtained during the stress test. The top line of Figure 1 is the record of a 60-cycle timing signal, the wide segments indicating the period of illumination of the glow lamp on the left; the narrow seg-

ments, that of the lamp on the right. The second line represents the eye movements; the third, the velocities of these movements; the fourth, the accelerations. Analysis of these records showed that there was no decrease in acceleration of the eyeball and, therefore, no reduction of the muscular forces during the stress test. Even after a stress period as long as 28 min. (Subject 10) the acceleration was found to be constant within the limits of the measuring accuracy.

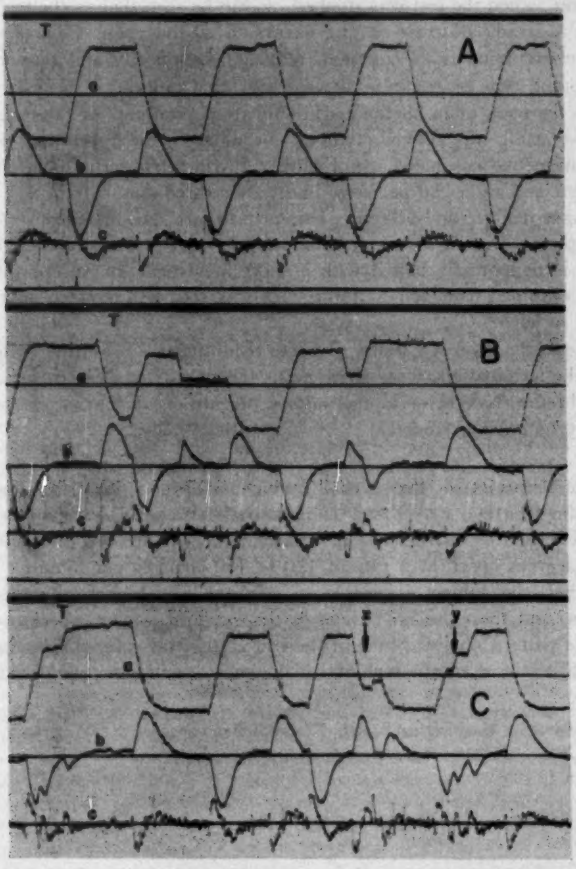

Fig. 2.—Record (Subject 2)) of excursion (*a*), velocity (*b*), and acceleration (*c*) of the eyeball under stress: row *A*, performance at start of test; row *B*, performance after three minutes; row *C*, performance after seven minutes. T is record of timing signal.

While no impairment of the muscular forces under stress was found, two main variations of the original pattern occurred during the stress test. The records show either a decreasing excursion of the eyes or the inability of the subject to move the eyes rhythmically and in step as desired.

The first of these changes is illustrated in Figure 1. The subject had been asked to fixate accurately the neon lamps during the test, and he usually terminated the test when he felt that he was unable to do this. However, the records indicate that the eyes were not moved the full 90 degrees, and thus no direct fixation was possible; the subject was apparently satisfied with an extrafoveal image. Such a response is shown in Figure 1, sample *A* being the performance at the start of the stress period; *B*, the performance after five minutes, and *C*, the performance after 10 min., when the test was stopped. It can be seen that as the test progressed there was a steady decrease in the excursion of the eyes. Coincident with the decrease in excursion is, of course, a reduction in velocity and acceleration.

The second type of response showed a general failure on the part of the eyes to keep up the pace, or to hit the mark. Apparent "stalling" or "freezing up," with no response at all, was noticed at times. In addition, the appearance of numerous "split" movements was noted. In Figure 2*A* symmetrical rhythmic lateral movements to left and right can be seen. In *B*, after three minutes, the eye movements are no longer definite and accurate, but, instead, are late in response, out of step, and incomplete. In *C*, after seven minutes, a movement from left to right is shown at *x*; this is temporarily halted with a short movement in the opposite direction and then continued from left to right. At *y* is seen a three-step split movement from right to left.

In all tests the subject's ability to fixate accurately was restored within 20 to 30 sec. after the termination of the stress test. There was no demonstrable difference in the calibration record, or in the pattern or rate of reading, after the period of rapidly alternating movements.

SUMMARY

The acceleration of the eyeball, which is proportional to the muscular force causing the eye movement, is measured with an electronic method. The subject is exposed to a stress test in which the eyes fixate two alternately flashing lights, 90 degrees apart, at a rate of 120 to 150 changes per minute. The test is stopped when the subject is unable to continue any longer. No decrement of acceleration, and therefore no fatigue of the extrinsic ocular muscles, was found. Instead, the pattern obtained toward the end of the test showed either a decreased excursion of the eyes or the inability of the subject to move the eyes rhythmically and in step.

The Illuminating Engineering Society Research Fund sponsored this research.

ALOPECIA ASSOCIATED WITH AN INTRAOCULAR FOREIGN BODY

Report of a Case

HARRY McGRATH, M.D.
BAY SHORE, N. Y.

COMPLETE baldness does not appear to be a credible complication of an injury to the eye. Changes in the hair of the scalp, brow, and lids, however, have frequently been observed in association with many diseases of the ocular tissues. It should, therefore, be interesting to present the case of a middle-aged man in whom alopecia of the scalp, brows, and lids developed a short time after a foreign body was extracted from the vitreous.

REPORT OF CASE

A man aged 30, whose general appearance was consistent with his age, was first seen on April 8, 1948. He was of dark complexion, with a head of thick, curly black hair (Fig. 1). His brows were heavy. Even before the serious nature of his injury was explained to him, he seemed a nervous type of person who was unduly depressed by his accident and apprehensive about treatment. A fragment of metal had pierced the cornea of the right eye and lodged in the vitreous about four hours previously. He was transferred to the New York Eye and Ear Infirmary, where the foreign body was removed by the anterior route by Dr. Conrad Berens. During his stay in the hospital there was no complication except the mild uveitis which followed the operation and the development of a traumatic cataract. Three weeks after the injury he left the hospital. The eye gradually became quiet, and when he was seen at the office two weeks later the cornea was clear except for the scar where the foreign body had penetrated. The anterior chamber was clear, and the iris was intact except where a small rent indicated the wound of entrance. The lens was opaque. Light projection was exact. The patient was despondent about the loss of vision, and he complained of insomnia and severe pain over the right brow, which extended upward as far as the vertex of the skull.

About six weeks later he noticed whitening and falling out of the cilia of the right eye. He seemed abnormally depressed. He was not seen again for about six months. When he returned, on Oct. 2, the change in his appearance was shocking. He had the appearance and carriage of a man in his sixties. There was almost complete absence of hair on the scalp, and the scanty remnants were thin and white. The eyebrows were pale and deficient. The few remaining lashes were thin and colorless. He could count fingers readily at a distance of 6 ft. (1.8 m.). The lens had completely absorbed, and there was a thin membrane with a clear central zone, through which details of the fundus could be seen with difficulty. The retina was apparently intact and grossly normal. The cornea and anterior chamber were clear. Tension was 22 m. Schiøtz.

He was referred for dermatological consultation. There was no abnormality of the hair of the axillas or the pubis. Scattered over the trunk were seven depigmented macules, but there was nothing to suggest vitiligo.

General physical examination revealed as significant points in the history, a continuous right-sided headache, constant thirst, and complete loss of libido. During his hospitalization and convalescence, drug therapy had been confined to preoperative and postoperative use of sedatives, atropine and ethylmorphine hydrochloride ("dionin"). Penicillin was the only antibiotic admin-

From the Ophthalmological Service, Southside Hospital.

istered. There was nothing to suggest that any therapeutic agent could have accounted for the alopecia. Nothing in the physical examination indicated any organic cause of the alopecia or the premature senility.

Laboratory studies were made, with the results noted:

Fig. 1.—Patient's appearance on April 8, 1948.

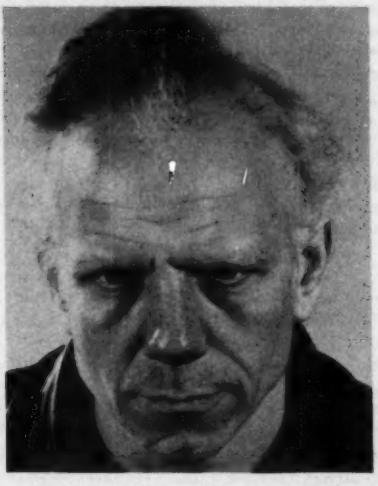

Fig. 2.—Patient's appearance in January 1951.

The urine was normal. The Mazzini test of the blood gave a negative result. The basal metabolic rate was + 2%. The serum cholesterol measured 120 mg., and the fasting blood sugar 92 mg., per 100 cc. The urinary excretion of 17-ketosteroids was 17.4 mg. a day. A complete blood count showed 5,400,000 red cells, 16 mg. of hemoglobin per 100 cc., and 9,000 white cells, with 61% neutrophils, 7% stab forms, 54% mature forms, 36% lymphocytes, and 3% eosinophils.

It was considered that the general physical condition, together with the results of the laboratory examinations, ruled out the possibility of any organic cause of the alopecia.

Neurological examination disclosed pronounced hyperesthesia over the whole area innervated by the first branch of the trigeminal nerve on the right. No abnormal neurological signs were elicited.

The patient was seen at irregular intervals until the last examination, on Feb. 1, 1951. At this time his mental condition was greatly improved, and he seemed much better adjusted,

Fig. 3.—Loss of cilia and eyebrows (January 1951)

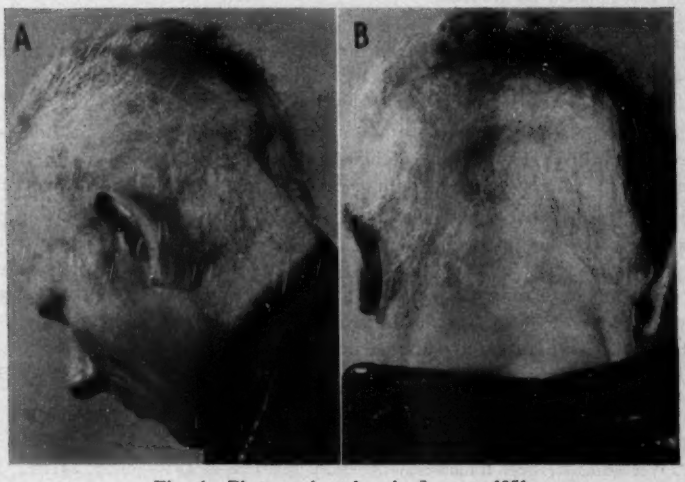

Fig. 4.—Photographs taken in January 1951.

possibly because the visual acuity in the right eye had improved considerably and the supraorbital pain was only occasionally evident. With correction, vision in the right eye was then 20/50. The eye was quiet, and the tension was 22 mm. (Schiøtz). The pupillary membrane was transparent. Details of the fundus could be clearly seen, and there was no abnormality. In the frontal region there had been a heavy regrowth of hair, but there were still large patches of alopecia on the vertex and over the occiput. The hair was coarse and dry. The eyebrows were almost nonexistent. Only a few thin, pale cilia were present on the lids of each eye. The beard was sparse and white. He found it necessary to shave only once a week (Figs. 2, 3 and 4).

COMMENT

The thorough physical examination in this case excludes the possibility of systemic or local infection, drugs, parasites, poisons, endocrine disturbance, or any of the other causes of alopecia cited by McCarthy [1] in his classification of the causes of alopecia. Since cranial trauma has often been followed by alopecia and poliosis, the possibility that the ocular injury in this case represented a localized area of cranial trauma requires consideration. As early as 1908, Wechselmann [2] made a thorough study of traumatic alopecia. He gave references to a large number of instances of head injuries which were followed at varying periods by loss of hair. None of these injuries seems to have involved the eye directly. A single case observed by Wechselmann himself, and discussed in detail by him, is representative.

A man aged 27 fell and injured the occipital region of his head. After 16 mo. the hair began to fall out in bunches all over his body and head. There was extensive alopecia areata of the scalp; the eyebrows had a few isolated dark hairs, but otherwise he did not have even lanuginous hair. The eyelashes on the left side were deficient. There was no beard. Wechselmann assumed that peripheral trophic disturbances were induced by a state of central irritation following trauma. He thought that psychic trauma, especially fright, produced a similar effect in some of the cases which he studied.

McCarthy included under the heading of alopecia neurotica the alopecia occurring in hysterical patients. He described a case in which alopecia developed after the sinking of a small boat. He quoted Kant as describing a disturbance of libido in three patients in whom alopecia developed.

Steindorf,[3] in 1914, discussed the occurrence of vitiligo of the lids and poliosis after blunt injury, but in none of his cases was any direct injury to the globe mentioned. Further reference to these cases of Steindorf's will be made later.

Hague [4] noted that depigmentation of the cilia and eyebrows following painful trauma (he did not mention whether this trauma involved the eye) and nontraumatic intraocular inflammation has long intrigued ophthalmologists. He cited the works of Arabian physicians [5] in the Middle Ages who reported canities in "thirty-seven common ocular disorders." Duke-Elder [6] says that depigmentation of the lashes with chronic uveitis, particularly sympathetic ophthalmitis, is an observation of considerable antiquity, dating back as far as Schenkl,[7] in 1873, and

1. McCarthy, L.: Diagnosis and Treatment of Diseases of the Hair, St. Louis, C. V. Mosby Company, 1940, p. 113.

2. Wechselmann: Über traumatische Alopecia, Deutsche med. Wchnschr. **34**:1982, 1908.

3. Steindorf, K.: Vitiligo der Lider und Poliosis nach stumpfer Verletzung, Klin. Monatsbl. Augenh. **53**:188, 1914.

4. Hague, E. B.: Uveitis; Dysacousia; Poliosis, and Vitiligo: Theory as to Cause, Arch. Ophth. **31**:520 (June) 1944; addendum **31**:562 (June) 1944.

5. Babel, J.: Syndrome de Vogt-Koyanagi, Schweiz. med. Wchnschr, **69**:1136, 1939; cited by Hague.[4]

6. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1941, Vol. 3, p. 2364.

7. Schenkl, A.: Fall von plötzlich aufgetretener Poliosis circumstripta de Wimpern, Arch. Dermat. u. Syph. **5**:137, 1873.

Jacobi,[8] in 1874. In 1908 Vogt[9] made his first report of a case of nontraumatic idiopathic uveitis accompanied with poliosis and alopecia. Koyanagi[10] published a paper in 1929 describing the disease in full and presenting 16 cases observed by himself and others. By this time vitiligo had been added as an essential component of the disease complex, which has since become known as the Vogt-Koyanagi syndrome.

Rosen[11] summarized the cases reported up to 1945 and found alopecia recorded in 31 of 40 cases of the disease. He quoted Rubino[12] as expressing the opinion that the uveitis is the result of infection with a virus having a specific uveotropism, while the same virus accounts for the pigmentary phenomena because of its specific melanotropism. He also considered the possibility of sensitivity to uveal pigment to account for the disease complex. Harada's disease is considered to be closely allied to, if not identical with, the same syndrome.[13]

Carasquillo,[14] in 1942, and Hague,[4] in 1944, have recently studied this syndrome in great detail, and their papers present complete bibliographies of the literature concerning the pigment changes associated with uveitis. Hague concluded that all the elements of the Vogt-Koyanagi syndrome were the result of a virus infection involving the hypothalamus. Carasquillo, in an exhaustive study of the syndrome, considered the possibility of anaphylaxis to uveal pigment, virus infection, and neurogenic factors.

Pisetsky and Kozinn[15] reported a case of total alopecia in a child of 10 yr. associated with pigmentary disturbances in the retina and optic nerve atrophy. They could offer no explanation for the association of alopecia with the ocular disorder and admitted that the cause remains obscure. Tomkinson[16] and Harford[17] noted the frequent occurrence of alopecia with strabismus. They gave no reason to account for the association.

There seems to be only one authenticated case in the literature of extensive alopecia following an injury to the eye without the development of sympathetic ophthalmia. This concerns a patient observed by Khavin[18]—a man aged 30 who suffered a perforation of the left globe with a piece of steel. Khavin described him as "nervous and overanxious about the state of his health." He presented

8. Jacobi, J.: Vorzeitige und acute Entfärbung der Wimpern beschränkt auf die Lider eines erkrankten Auges, Klin. Monatsbl. Augenh. **12**:153, 1874.

9. Vogt, A.: Frühzeitiges Ergrauen der Zilien und Bemerkungen über den sogenannten plötzlichen Eintritt dieser Veränderung, Klin. Monatsbl. Augenh. **44**:228, 1906.

10. Koyanagi, Y. S.: Alopecia und Poliosis bei schwerer Uveitis nicht traumatischen Ursprungs, Klin. Monatsbl. Augenh. **82**:194, 1929.

11. Rosen, E.: Uveitis with Poliosis, Vitiligo, Alopecia and Dysacousia (Vogt-Koyanagi Syndrome), Arch. Ophth. **33**:281 (April) 1945.

12. Rubino, A.: Uveo-meningitisches Syndrom (Beitrag zum Studium der Harada-Krankheit), Ophthalmologica **101**:321, 1941; cited by Rosen.[11]

13. Cowper, A. R.: Harada's Disease and Vogt-Koyanagi Syndrome, Arch. Ophth. **45**:367 (April) 1951.

14. Carasquillo, H. F.: Uveitis with Poliosis, Vitiligo, Alopecia, and Dysacousia, Arch. Ophth. **28**:385 (Sept.) 1942.

15. Pisetsky, J. E., and Kozinn, P. J.: Total Alopecia Associated with Ocular Disorders, Am. J. Dis. Child. **64**:80 (July) 1942.

16. Tomkinson, J. G.: Alopecia Areata and Strabismus, Brit. M. J. **2**:518, 1924.

17. Harford, C. F.: Alopecia Areata and Strabismus, Brit. M. J. **2**:643, 1924.

18. Khavin, A. O.: Alopecia in Traumatic Iridocyclitis, Vestnik oftal. **17**:140, 1940.

"dermatographism" and was abnormally depressed. There was a traumatic cataract, which was subsequently removed, and the eye became quiet. There does not seem to have been any foreign material in the globe. Three weeks after the injury the hair began to fall out, and within a month only a small tuft of hair remained on the temples on each side. Three months later there was a normal growth of hair on the scalp, with no change in color or texture. Khavin also gave an incomplete report of a similar case in which there was some loss of hair after a perforating injury. He did not refer to the opposite eye. Moutier and Legrain [19] mentioned an ocular injury, described as "a wound of the left eye," with loss of hair three weeks later. "Ocular radiotherapy" had been used, but it was applied only locally, and the authors saw no connection between this treatment and the alopecia.

Cramer's [20] case is similar to that described by Khavin and the one presented in this paper with the important distinction that in his case sympathetic ophthalmitis developed. Cramer stated that he had never been able to accept the idea of the anaphylactic genesis of sympathetic ophthalmia until his doubts were dispelled by his observation of the following case:

A woman aged 38 had a traumatic luxation of the lens. The lens was removed at operation, and the eye healed without immediate complications. Later, sympathetic inflammation developed in the opposite eye. Three months after the onset of the uveitis, the hair, which had been heavy at her last visit, was noticeably thinner. Two months later she was almost bald, and the remnants of hair were white. Only a few lashes remained. The axillary and pubic hairs were also thin and white. In view of the dissemination of the hair disease over the whole body, Cramer suggested that the disease picture was based on anaphylaxis.

It is interesting to conjecture that, in the quite likely event of sympathetic ophthalmitis in the case which I have outlined, the alopecia would have been considered to lend support to the theory of anaphylaxis. But as only one eye was involved, it is obvious that what Cramer considered an anaphylactic phenomenon can occur without any consideration of sensitivity to uveal pigment. Steindorf,[3] whose observations on vitiligo of the lids and poliosis following blunt injury to the eye were previously referred to, mentioned this case of Cramer's specifically. He considered the interpretation of the symptom complex as a manifestation of anaphylaxis a mere conjecture, since it does not apply to the hair changes which he described. In a case which he cited, a patient of Cassirer's,[3] a young girl, after severe exertion and fright, exhibited grayness of the eyebrows and lashes in association with Horner's syndrome. As not only sympathetic disturbances but also trophic anomalies were present, the case is of special interest in calling attention to the possible neurogenic origin of the pigment and hair changes observed in association with diseases of the eye. Steindorf assumed that in cases of head injury the trophic nerves of the scalp are injured, with consequent vascular disturbances in the region and alteration of pigment supply. This nutritional disturbance leads to shriveling of the hair cells.

19. Moutier, F., and Legrain, P.: La pelade traumatique, Ann. dermat. et syph. **9**:268, 1928.

20. Cramer, E.: Zur Frage der anaphylaktischen Entstehung der sympathischen Entzündung, Klin. Monatsbl. Augenh. **51**:205, 1913.

Galewsky [21] was of the opinion that alopecia neurotica, or alopecia traumatica, is a special type of baldness and should be distinguished from alopecia areata. Alopecia traumatica is a form of baldness following injury to the head, neck and face; shock; fright; worry, etc. The case presented here seems to fit into this classification. It would, therefore, appear that there was no specific connection between the uveal injury and the alopecia. The local disturbance in the eye can be assumed to have initiated in an unknown manner through the nervous system some derangement of the trophic nerves of the scalp and face. As both my patient and the one observed by Khavin were of a neurotic type, it may be that some inherent disposition is necessary. Some such trophoneurotic atrophy could account for the hair changes seen in sympathetic ophthalmia and the Vogt-Koyanagi syndrome and allied diseases without the postulation of a virus infection or anaphylaxis to uveal pigment.

SUMMARY

Depigmentation and loss of hair in the scalp, lids, and brows have been known for many years to occur with disease of the eye. These phenomena have been especially noted in connection with the Vogt-Koyanagi syndrome and sympathetic ophthalmia. A case of alopecia of the scalp, brows, and cilia followed the removal of an intraocular foreign body. There are many references in the literature to the development of alopecia subsequent to cranial and facial trauma, but instances of this bizarre complication after an ocular injury are extremely rare.

The hair changes in the case presented seem to be manifestations of alopecia traumatica caused by cranial injury in a subject of neurotic disposition. The fact that such changes can occur after unilateral traumatic uveitis may throw light on the origin of similar changes in the bilateral nontraumatic idiopathic uveitis, known as Vogt-Koyanagi disease and in sympathetic ophthalmia.

Dr. Frank Bussey, Dr. Joseph Singer, Dr. Lothar Kalinowsky, and Dr. Joseph Lambert, of the Southside Hospital staff, assisted in the examination of the patient.

12 Shore Lane.

21. Galewsky, E.; quoted by McCarthy, L.: Histopathology of Skin Diseases, St. Louis, C. V. Mosby Company, 1931, p. 148.

RATE OF FLOW OF AQUEOUS HUMOR IN NORMAL AND SCORBUTIC GUINEA PIGS

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THE INTIMATE mechanism for the secretion of aqueous humor and the part played by ascorbic acid in this process are still unknown. Friedenwald and collaborators [1] proposed a secretory mechanism in which ascorbic acid is thought to play an important part as a metabolic mediator in a "redox-pump" system. In support of this hypothesis, several interesting observations have been presented. Of these, two seem to have the most direct bearing on the question: In even moderate scurvy, there are already a marked slowing in the rate of transport of basic dyes from stroma to epithelium in the ciliary processes and, simultaneously, a pronounced reduction in the rate of refilling of the anterior chamber after partial evacuation. This was thought to indicate a reduction in the rate of secretion of aqueous. The fact that the intraocular pressure was not disturbed was explained by the assumption of a stabilizing mechanism. Both these effects of scurvy were almost immediately reversible when ascorbic acid was supplied.

At the time of these studies of Friedenwald and collaborators there was no satisfactory method of measuring the rate of secretion of aqueous in intact eyes. Since the rate of refilling of an evacuated anterior chamber could possibly depend on factors other than rate of secretion of aqueous, the following experiments were carried out to reinvestigate the matter, advantage being taken of the new method for measuring aqueous flow developed by Bárány and Kinsey.[2]

From the Pharmacological Institute, University of Uppsala.

This investigation was supported by a grant from the Swedish Medical Research Council. The work was presented in part at the Eighteenth International Physiological Congress, Copenhagen, August, 15-18, 1950.

1. Friedenwald, J. S., and Stiehler, R. D.: Circulation of Aqueous: A Mechanism of Secretion of the Intraocular Fluid, Arch. Ophth. **20**:761 (Nov.) 1938. Friedenwald, J. S.; Buschke, W., and Michel, H. O.: Role of Ascorbic Acid (Vitamin C) in the Secretion of the Intra-Ocular Fluid, Tr. Am. Ophth. Soc. **37**:310, 1939; Role of Ascorbic Acid (Vitamin C) in Secretion of Intraocular Fluid, Arch. Ophth. **29**:535 (April) 1943.

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METHODS

The rate of flow of aqueous was calculated from the rate of disappearance from the aqueous and the steady-state distribution ratio of paraaminohippuric acid in the aqueous and plasma, in essentially the same manner as in the rabbit experiments, previously described by Bárány and Kinsey. Only the deviations from the technique described for rabbits are given below.

Unanesthetized male guinea pigs, weighing 400 to 800 gm., were used. Controls were kept on a diet of water, hay, oats, and turnips ad libitum. Two scorbutogenic diets were used. The first diet (A) consisted of 45 parts bran, 25 parts crushed oats, and 30 parts dried whole milk. Every fifth day the animals were given 1 ml. of cod liver oil. The constituents of this diet had not been heated, and there probably was a small amount of ascorbic acid in the milk powder. In a second series severer scurvy was aimed at, and the diet (B) was as follows: 24 parts bran, 50 parts crushed oats, 15 parts dried skim milk, 10 parts filtered butter fat, and 1 part sodium chloride. The bran, oats, and milk powder were heated for two hours at 110 C., with free access of air.

Injections were made into a vein of the hindleg. Continuous subcutaneous infusions were given at constant rates by means of motor-driven syringes. Samples of aqueous were taken after topical anesthesia with lidocaine and luxation of the eye. (The eye of the guinea pig can be luxated without much difficulty by means of a small spoon-shaped instrument.) The samples of aqueous were immediately blown down into rubber-stoppered tubes and weighed. The specific gravity was taken as 1.00, involving an error of less than 1%. The amount of aqueous obtained was usually 20 to 40 mg. The few eyes which were used twice were checked with the slit-lamp microscope.

Paraaminohippuric acid was determined colorimetrically according to the method of Smith and associates.[3] The readings were taken in microcuvettes of 5-cm. length in a Zeiss *Stufen-Photometer,* filter S53. The topical anesthetic used gives no color with the reagents, nor do blood and aqueous from animals which have not received injections.

Ascorbic acid was determined by placing the aqueous from a single eye into 0.1 ml. of 4% metaphosphoric acid and titrating with dichlorophenolindophenol sodium. A magnetic stirrer and the use of Aglä microsyringes as burettes greatly facilitated the titration.

In the experiments intended to yield rates of disappearance of the test substance, the animals received intravenously 0.4 gm. per kilogram of paraaminohippuric acid as a neutralized 10% solution. The drug was injected rapidly. No toxic symptoms were discernible. Blood and one aqueous sample were taken after 90 min.; the other aqueous sample and a second blood sample were usually taken 60 min. later.

In the experiments intended to yield steady-state ratios, the animals were fixed on a board and kept warm by an electric pad. They were given a 5% solution subcutaneously at a rate of about 1.3 ml. per hour for six hours. At this low rate of injection there seems to be almost complete absorption from the site of injection in most cases; the blood concentration curves were fairly level. In some experiments, however, absorption seemed to be erratic, and in the later experiments a small amount of a hyaluronidase preparation from bull testis was added to the infusion fluid. The blood curves in these cases were as level as in rabbits with continuous intravenous infusion. Samples of blood were taken 1.5, 3, 4, 5, and 6 hr. after the start of the infusion. Samples of aqueous were taken in both eyes at six hours.

The water content of three plasma samples from different control animals, determined by drying for three hours at 105 C., was found to be 93, 94, and 94%.

The amount of freely diffusible paraaminohippuric acid was determined by dialyzing plasma against 0.9% sodium chloride in cellophane bags at two concentration levels of paraaminohippuric acid, 0.01 and 0.001%. Three samples gave an average diffusible fraction of 81% of the higher, and 78% at the lower, concentration. The concentrations used are approximately those present in the animal experiments.

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The mathematical methods used were exactly the same as those previously described for rabbits. Because of the possible occurrence of highly erratic values, medians, instead of the more conventional means, were used.

RESULTS

Ascorbic Acid in Aqueous During Development of Scurvy.—Table 1 summarizes results obtained with animals on the less severe diet (A). In the controls, the mean concentration of reducing substance was 8.2 mg., calculated as ascorbic acid, per 100 ml. This is not the saturation level, but the control animals grow and reproduce on the diet without difficulty. It is seen in Table 1 that after eight days on Diet A there was no further decrease in reducing power. It is not probable that the value 1.3 to 1.4 mg. per 100 ml., found in the aqueous of these animals, really represents ascorbic acid. It may well be caused, partly at least, by other reducing substances. This was evident, too, from a small series, of seven animals, on Diet B for 11 to 14 days, which at the time were quite ill from scurvy. The following concentrations, in milligrams of "ascorbic acid" per 100 ml., were found, the figures in parentheses indicating the number of days on Diet B: 0.7; 0.7; 0.6; 1.7 (11); 1.3; 1.9 (13); 0.6 (14). With these results as a guide, the main series of experiments with paraaminohippuric acid were done after the animals had been 10 days on Diet A. At this time, the animals still appeared fairly healthy. This observation agrees well with that of Friedenwald and associates, who found that severe depletion of the ascorbic acid of the aqueous occurred before any other clinical sign of scurvy was detectable.

TABLE 1.—*Concentration of Reducing Substances in the Aqueous During Development of Scurvy*

No. of Days on Diet A	No. of Eyes from Different Animals	Ascorbic Acid, Mg./100 Ml. (Av.)
0	16	8.2
2	4	5.9
3	4	2.8
4	8	4.4
8	6	1.4
10	7	1.3

Steady-State Aqueous-Plasma Distribution Ratio of Paraaminohippuric Acid in Normal and Moderately Scorbutic Animals.—Table 2 summarizes the results of 10 experiments on normal animals and of 15 experiments on scorbutic animals, which had been on Diet A for 10 days. The distribution ratios (R) were calculated from the concentrations observed, using equation 5 in Bárány and Kinsey [2b] and a value for k_{out} of 0.017 for the normal eyes and 0.016 for the scorbutic eyes. All the distribution ratios of this table are uncorrected for plasma binding of paraaminohippuric acid and for the water content of plasma. The values obtained for scorbutic animals, 0.15, and for normal animals, 0.17, do not differ much and are closely similar to values for rabbits and chickens. It is interesting to note that if there had been an increased capillary permeability in the iris, as a result of the scorbutic state, the value for the scorbutic animals would have been higher. Now, it actually is insignificantly lower. It would have been better if both groups of

animals had belonged to the same weight group, but body weight does not seem to influence the results. There is no correlation with weight discernible in either group.

Paraaminohippuric-acid concentrations are listed only for one eye in some cases of the scorbutic group, because ascorbic-acid determinations were made on the opposite eye. The values obtained are given in Table 2. There seems to be no correlation between these values and the distribution ratios.

TABLE 2.—*Plasma and Aqueous Levels of Paraaminohippuric Acid During Steady Subcutaneous Infusion of About 1.3 Ml. per Hour of 5% Solution in Normal and in Scorbutic Guinea Pigs**

		Paraaminohippuric Acid									
		Plasma, Mg./100 Ml.					Aqueous at 300 Min.		Steady-State Distribution Ratio (R)		Aqueous "Ascorbic Acid," Mg./100 Ml.
		Time of Sample, Min.									
Animal	Weight, Kg.	90	180	240	300	360	Left	Right	Left	Right	
					Normal Controls						
12	0.50	7.3	8.1	9.2	11.6	8.1	1.4	1.6	0.15	0.17	...
13	0.50	6.5	6.4	6.9	5.5	3.3	0.7	1.0	0.14	0.21	...
14	0.40	7.1	6.1	7.0	6.2	6.5	0.9	1.1	0.14	0.17	...
15	0.48	12.0	8.1	10.5	10.5	8.5	1.6	1.6	0.17	0.17	...
17	0.50	12.4	17.5	13.4	12.7	13.4	3.1	2.1	0.23	0.16	...
18	0.50	10.2	6.0	10.2	12.3	15.3	2.4	1.6	0.19	0.12	...
19	0.50	7.1	10.8	9.2	8.9	8.5	1.9	1.4	0.22	0.15	...
20	0.55	1.9	6.8	6.8	6.8	7.2	1.1	0.9	0.17	0.13	...
21	0.55	5.8	6.1	5.8	5.1	5.8	1.2	1.1	0.21	0.20	...
22	0.53	2.2	2.7	6.2	7.9	7.5	1.6	1.5	0.23	0.20	...
Mean:	0.50								Median, each eye a unit: 0.17 Median, each animal a unit: 0.17		
					Scorbutic Animals						
22	0.75	10.9	13.2	11.0	12.4	12.7	1.2	2.5	0.10	0.21	...
28	0.70	13.6	11.4	10.6	9.8	10.5	...	1.4	....	0.14	...
1	0.67	7.6	12.7	11.6	10.8	10.8	1.2	1.6	0.11	0.15	...
29	0.76	6.4	7.7	7.9	7.3	7.0	1.8	2.1	0.25	0.30	...
30	0.81	9.1	8.3	8.1	7.9	8.0	1.3	2.6	0.17	0.33	...
16	0.71	7.7	8.3	9.8	9.4	9.1	1.6	1.8	0.17	0.20	...
17	0.70	7.5	8.0	8.1	8.3	8.0	1.4	0.8	0.18	0.10	...
3	0.82	5.4	10.1	9.8	12.7	12.8	1.1	1.8	0.10	0.16	...
7	0.88	5.4	7.1	6.3	9.2	7.9	1.8	...	0.22	....	1.3
9	0.80	5.3	5.8	8.0	9.5	12.8	1.4	...	0.14	....	1.3
27	0.81	3.3	5.5	6.7	7.0	8.4	1.1	...	0.16	....	1.3
34	0.60	4.6	5.5	5.8	6.3	6.3	...	1.3	....	0.21	1.3
36	....	5.0	6.3	5.1	9.6	14.3	...	1.0	....	0.09	...
10	0.81	7.5	7.1	7.2	7.3	7.2	...	2.6	....	0.37	2.5
38	0.52	7.4	7.5	9.8	10.1	10.5	1.3	...	0.13	....	2.4
Mean:	0.74								Median, each eye a unit: 0.19 Median, each animal a unit: 0.15		

* All times are calculated as minutes after start of the infusion. All concentrations are given in milligrams per 100 ml.

Rate of Disappearance of Paraaminohippuric Acid from Aqueous in Normal and in Scorbutic Animals.—Moderately Scorbutic: Using the distribution ratios 0.17, for normal, and 0.15, for scorbutic animals, arrived at in the preceding section, the true rate of disappearance of paraaminohippuric acid from the aqueous can be calculated from directly observed values according to equation 4 of Bárány and Kinsey.[2b] Table 3 gives the results of 23 experiments on the rate of disappearance in controls and the same number of experiments in scorbutic animals who had been on Diet A for 10 days. In the first three animals of the normal group, the interval between the taking of the first and the second pair of samples was 30 min. In all later experi-

ments it was about 60 min. It is seen from Table 3 that the rate of disappearance (k_{out}) is not lower for the scorbutic animals. In most cases the correction, that is, the difference between true and apparent rates of disappearance, is small or moderate, indicating that there was only a low plasma level of paraaminohippuric acid during the interval between the taking of the aqueous samples. In some cases, however, there was a higher plasma level, and here the correction sometimes is rather high. Since the formula used for correction, while probably satisfactory on an average, is nonetheless based on assumptions which might not be justified in the single case, such values as contain a large correction term are slightly suspicious. If values in which the "true" value exceeds the apparent value by more than 40%

TABLE 3.—*Rate of Disappearance of Paraaminohippuric Acid from Aqueous in Normal and in Scorbutic Guinea Pigs, Expressed as Per Cent per Minute of Contents of Anterior Chamber*

Normal Controls				Scorbutic Animals			
Animal	Weight, Kg.	Apparent Rate of Disappearance	Corrected Rate of Disappearance: $100 \cdot k_{out}$	Animal	Weight, Kg.	Apparent Rate of Disappearance	Corrected Rate of Disappearance: $100 \cdot k_{out}$
3	0.40	1.3	1.4	4	0.83	1.2	1.4
4	0.40	1.8	2.7	5	0.74	2.4	2.6
5	0.34	1.5	1.8	11	0.88	1.1	3.2
6	0.45	1.3	1.5	12	0.73	0.5	0.9
7	0.50	1.5	1.9	13	0.83	0.9	1.4
8	0.45	1.0	1.2	14	0.88	1.7	2.1
9	0.48	1.0	1.0	23	0.75	1.5	1.6
10	0.80	0.4	0.5	30	0.73	1.0	2.6
11	0.48	0.9	1.1	31	0.68	1.7	1.9
23	0.74	0.5	0.7	28	0.80	0.9	1.4
24	0.67	1.7	3.2	37	0.70	2.0	2.2
25	0.72	0.6	0.7	29	0.75	1.6	1.7
26	0.65	1.4	2.1	40	0.61	1.3	1.5
27	0.53	1.4	1.6	41	0.68	1.6	1.8
34	0.68	1.7	3.0	42	0.57	1.2	1.5
35	0.60	1.8	3.2	43	0.65	3.0	3.4
36	0.60	2.2	2.4	44	0.72	1.6	1.7
37	0.59	1.3	1.4	45	0.55	1.4	1.7
38	0.66	1.6	2.1	46	0.47	1.7	1.9
39	0.59	2.1	2.5	47	0.50	2.0	2.4
40	0.81	1.3	1.4	48	0.48	1.7	1.9
41	0.65	1.4	1.7	49	0.47	2.6	2.8
42	0.71	1.7	1.9	50	0.43	2.2	2.3
Mean:	0.59	Median: 1.4	Median: 1.7 Mean: 1.8 ± 0.2	Mean:	0.67	Median: 1.6	Median: 1.9 Mean: 2.0 ± 0.1

are discarded (Cases 4, 10, 23, 24, 26, 34, and 35 in the control group and 11, 12, 13, 30, and 33 in the scorbutic group), the median for the control group is shifted from 1.7% to 1.55% per minute, while the median for the scorbutic group, 1.9% per minute, is not shifted at all. The average values can, therefore, be viewed with considerable confidence, in spite of the approximations committed in the calculations.

The results obtained so far do not indicate any reduction in the rate of flow in moderately scorbutic animals. The rate of flow, as shown by Kinsey and Bárány,[2c] lies between the limits k_{out} and $(1\text{-}R_1) \cdot k_{out}$. In this formula, R_1 is the steady-state distribution ratio (R) after correction for plasma binding of test substance and for the water content of the plasma. In normal guinea pigs the water content of plasma is 93 to 94%, and the bound paraaminohippuric acid, about

20%. This makes $R_1 = 1.2R$. Since the present experiments have shown R, as well as k_{out}, to be about the same in the scorbutic group as in the control series, the rate of flow of aqueous cannot be very different in the two groups of animals, even in the unlikely case that considerable differences between the groups were to be found in the water content or the paraaminohippuric acid-binding ability of the plasma. This possibility has not been investigated. A further remote possibility is that considerable differences in the volume of the anterior chamber exist between the two groups. Since flow is calculated as a fraction of this volume, a constant flow fraction in the face of such differences would indicate differences in absolute rate of flow. This possibility can be ruled out, however. The mean amount of aqueous obtained in the control group was 36 cu. mm. per eye, and that in the scorbutic group, 37 cu. mm. Eeven if the whole amount in the anterior chamber is not recovered on puncture, there is no reason that differences in yield should exist between the groups.

Severely Scorbutic: The scorbutic group in the preceding section consisted of animals with only moderate scurvy. In order to see whether a diminution of flow

TABLE 4.—*Plasma and Aqueous Levels of Paraaminohippuric Acid in Severely Scorbutic Guinea Pigs After Six Hours of Continuous Subcutaneous Infusion of About 1.3 Ml. per Hour of 5% Solution**

Animal	Weight, Kg.	Days on Diet B	Plasma	Aqueous, Left Eye	Ratio Aqueous: Plasma	"Ascorbic Acid," Right Eye
53	0.64	11	9.4	1.7	0.19	0.6
54	0.58	11	6.8	1.2	0.18	1.7
55	0.64	11	8.3	0.8	0.10	...
56	0.68	13	9.6	1.7	0.18	1.3
57	0.62	14	9.1	2.2	0.24	0.6
58	0.64	18	8.7	1.4	0.16	1.9
Mean:	0.63				Median: 0.18	

* All values are given in milligrams per 100 ml.

could be demonstrated in animals with severer scurvy, a number of guinea pigs were placed on Diet B for 11 to 18 days. At the end of this period they were in poor condition from scurvy, and in selecting the animals for the day's experiments one tended to use those first which looked bad and threatened to die if not used soon. Several animals died during the experiments.

It was clearly impossible to subject these animals to repeated heart punctures, as in the steady-state experiments conducted with the other groups. The procedure had to be simplified. The animals were given the subcutaneous infusion in exactly the same manner as the other groups, but only one sample of blood was taken, after six hours of infusion, at the same time as the sample of aqueous. Table 4 shows the results obtained in six steady-state experiments of this kind. In all cases the aqueous from the right eye was analyzed for ascorbic acid.

It is seen that at the end of the infusion there was an aqueous-plasma ratio for paraaminohippuric acid of 0.18. Since this is not directly comparable with the distribution ratios (R) of Table 2, the corresponding ratios of the mean aqueous concentration of the two eyes and the last sample of plasma have been calculated for the control animals of Table 2. The following series of ratios was obtained: 0.13, 0.14, 0.15, 0.19, 0.19, 0.19, 0.19, 0.20, 0.21, and 0.26, with a median of 0.19.

A comparison with the median for the severely scorbutic group, 0.18, shows that there is no significant difference. It is probable, therefore, that the steady-state distribution ratio, even in these deeply scorbutic animals, is about normal—possibly slightly subnormal. Since the normal value of R is 0.17, a value of 0.16 has been assumed in computing the corrections for the rate-of-disappearance experiments of the severely scorbutic group. The results of 10 experiments in this group are shown in Table 5.

As seen in Table 5, the experiments were all rather successful in so far as the necessary corrections have been small. The median value for the corrected rate of disappearance, 1.4% per minute, is somewhat smaller than the median for the normal controls, 1.7; but in view of the smallness of the group with severe scurvy, it is doubtful whether the difference is significant. The experiments show clearly, however, that the reduction in rate of flow of aqueous in cases of severe scurvy cannot be great. If there is any reduction, it is, moreover, quite doubtful whether

TABLE 5.—*Rate of Disappearance of Paraaminohippuric Acid from Aqueous in Severely Scorbutic Guinea Pigs, Expressed as Per Cent per Minute of Contents of Anterior Chamber*

Animal	Weight, Kg.	Days on Diet B	Apparent Rate of Disappearance	Corrected Rate of Disappearance: $100 \cdot k_{out}$
59	0.59	11	1.1	1.3
60	0.80	11	1.1	1.2
61	0.67	12	1.0	1.0
62	0.58	11	0.9	1.0
63	0.94	13	0.5	0.5
64	0.55	13	1.3	1.5
65	0.87	16	2.1	2.2
66	0.60	18	1.2	1.5
67	0.52	16	1.8	1.9
68	0.72	16	1.8	2.0
Mean:	0.68		Median: 1.15	Median: 1.4

it is specific. These animals were in very poor condition, and it would not be surprising if several functions were depressed.

Saturation with Ascorbic Acid.—The results of the preceding sections clearly do not agree with the observations of Friedenwald and collaborators made by other methods and thought to indicate greatly reduced formation of aqueous in scurvy. When informed of the discrepancy, Dr. Friedenwald pointed out that dietary differences could exist between guinea pigs in Baltimore and Uppsala and that his animals had received a fortifying dose of ascorbic acid the day before the experiment. In order to test whether the observed lack of difference between our controls and the scorbutic animals could be caused by a prescorbutic state of the controls, a series of experiments were performed on animals saturated with ascorbic acid.

The animals who were kept on the stock diet were given a subcutaneous dose of 50 mg. of ascorbic acid daily for at least two days before the experiment. A further dose of 50 mg. was given two hours before the start of the experiments on the rate of disappearance and at the start of the steady-state experiments. In some animals, 1 drop of one aqueous sample was taken for reduction titration. In all other respects, the experiments were performed in exactly the same manner as

those the results of which are presented in Tables 2 and 3. The results for these ascorbic-acid-saturated animals did not differ significantly from those for the other groups, as seen in Tables 6 and 7.

Table 6 shows that the steady-state distribution ratio, 0.14, is very nearly the same as that for the scorbutic group of Table 2 (0.15), while the ascorbic-acid

TABLE 6.—*Plasma and Aqueous Levels of Paraaminohippuric Acid During Steady Subcutaneous Infusion of About 1.3 Ml. per Hour of 5% Solution in Guinea Pigs Saturated with Ascorbic Acid**

		Paraaminohippuric Acid									
		Plasma					Aqueous at 300 Min.		Steady-State Distribution Ratio (R)		
		Time of Sample, Min.									Aqueous "Ascorbic Acid"
Animal	Weight, Kg.	90	180	240	300	360	Left	Right	Left	Right	
79	0.69	8.6	8.3	8.6	6.9	7.7	1.2	1.2	0.15	0.18	....
80	0.78	6.3	6.1	7.5	7.4	6.8	1.2	0.9	0.18	0.13	....
82	0.69	6.7	6.9	8.3	8.6	9.7	2.4	0.8	0.27	0.09	....
83	0.75	3.8	4.3	5.1	5.0	5.6	0.8	0.7	0.15	0.13	....
84	0.70	3.6	4.5	4.4	3.8	4.9	0.6	0.3	0.13	0.07	24.1
85	0.80	7.7	6.3	6.4	6.7	5.6	0.6	0.5	0.10	0.07	26.1
86	0.73	6.1	6.2	5.9	5.3	5.6	0.6	0.6	0.10	0.11	24.3
88	0.82	4.4	5.5	4.7	5.2	5.3	0.8	0.8	0.16	0.15	29.0
89	0.65	6.3	6.6	6.1	6.3	8.1	1.2	0.8	0.18	0.12	25.4
90	0.65	6.0	5.6	5.5	5.5	5.8	0.5	0.3	0.09	0.06	30.0
Mean:	0.72								Median, each eye a unit: 0.13		
									Median, each animal a unit: 0.14		

* All concentrations are given in milligrams per 100 ml. All times are given as minutes after start of the infusion.

TABLE 7.—*Rate of Disappearance of Paraaminohippuric Acid from the Aqueous in Guinea Pigs Saturated with Ascorbic Acid*

Animal	Weight, Kg.	Apparent Rate of Disappearance	Corrected Rate of Disappearance: $100 \cdot k_{out}$
91	0.65	1.3	1.5
92	0.65	1.3	1.9
94	0.79	0.9	1.0
95	0.60	1.7	1.9
96	0.57	1.5	1.9
98	0.50	2.5	3.1
99	0.55	1.7	2.1
100	0.56	1.7	2.2
102	0.62	0.9	1.2
103	0.64	1.6	1.8
	Mean: 0.61	Median: 1.5	Median: 1.9

value of the aqueous is at least 10 to 20 times as high. Table 7 shows that the median rate of disappearance, 1.9% per minute, is the same for the saturated group as for the scorbutic group. The close coincidence is fortuitous but shows that no considerable difference can exist between the groups.

Rate of Flow of Aqueous in the Guinea Pig.—Table 8 summarizes the results from the preceding sections and gives the rates of flow calculated from the R and k_{out} of the various groups. In these calculations, the water content of plasma has

been assumed uniformly to be 94%, and the percentage of freely diffusible paraaminohippuric acid has been taken as 80. It is seen that the "flow" column gives two limits for the rate of flow. These limits are not statistical limits of confidence but are set because of our lack of definite knowledge regarding the mode of entrance of paraaminohippuric acid into the anterior chamber. This does not imply, however, that all values within the interval are equally probable. As pointed out by Kinsey and Bárány [2c] and lately elaborated by Kinsey,[4] the fact that a number of different substances are brought into the anterior chamber of the rabbit by active transport shows that the active transport mechanism is highly unspecific. It is probable that it consists simply of the unidirectional flow of fluid through the space between iris and lens. If this should be true, the test substances, too, will probably enter mainly by means of this flow mechanism, and the true values for aqueous flow will be near the upper limits given in Table 8.

If it is true, however, that the test substance enters the anterior chamber mainly or predominantly by bulk flow from the posterior chamber, the validity of our formulas for calculating flow from the experimental data must be reconsidered. The

TABLE 8.—*Rates of Flow of Aqueous in Normal and Scorbutic Guinea Pigs**

	Steady-State Experiments			Rate of Disappearance Experiments			Rate of Flow	
Group	No. of Animals in Group	Distribution Ratio (R)	Aqueous "Ascorbic Acid"	No. of Animals	Apparent Rate of Disappearance	Corrected Rate: $100 \cdot k_{out}$	Assuming No Diffusion Through Iris	Assuming Maximal Diffusion Through Iris
Saturated	10	0.14	24-30	10	1.5	1.9	1.9	1.6
Control	10	0.17	8.2 (mean)	23	1.4	1.7	1.7	1.4
Moderate scurvy	15	0.15	1.3-2.5	23	1.6	1.9	1.9	1.6
Severe scurvy	6	0.16	0.6-1.9	10	1.2	1.4	1.4	1.1

* All rates are expressed as per cent per minute of contents of anterior chamber; concentrations, in milligrams per 100 ml.

theory upon which our mathematical calculations are based is a one-chamber theory. It assumes that the rate of entrance of substances by means of active transport into the anterior chamber is proportional to their instantaneous concentration in the plasma water. This is an oversimplification. A better approximation would be that they enter at a rate which is proportional to their concentration in the posterior chamber. The difference between the two approximations cannot be great, however. The posterior chamber has a small volume, which is rapidly turned over by the existent flow, and a relatively large contact surface with the blood. The fluid will therefore reach a condition of steady state very rapidly, and even if the blood concentration changes quickly, the concentration in the posterior chamber will follow with only a small lag. This lag is obviously unimportant, except possibly in experiments on the rate of disappearance. Here the lag will cause the concentration of paraaminohippuric acid in the posterior chamber to be slightly higher than the steady-state value corresponding to the instantaneous plasma level. Now, our corrections of the apparent rate of disappearance are designed to take into account replacement of test substance from the blood. It would be better if they

4. Kinsey, V. E.: A Unified Concept of Aqueous Humor Dynamics and Maintenance of Intraocular Pressure: An Elaboration of the Secretion-Diffusion Theory, Arch. Ophth. **44**:215 (Aug.) 1950.

were able to take into account replacement from the posterior chamber; but since posterior-chamber concentrations are unavailable, we have to be content with plasma concentrations. The latter are somewhat lower, and therefore our corrections are somewhat too small. The corrected rates of disappearance will, therefore, be slightly too low. It is probable, but not proved, that the error is negligible.

In order to test the adequacy of the corrections for replacement of test substance, Bárány and Kinsey [2b] prepared correlation diagrams of k_{out} against the ratio C_{pl}/C_{aq} at the time of taking of the first sample. These scattergrams seemed to indicate adequate correction. It is questionable, however, if the use of C_{pl}/C_{aq} is permissible. Since the aqueous concentrations are already decreasing when the first sample is taken, the concentration in this sample will be influenced by the rate of disappearance, k_{out}. Thus, the same variable, k_{out}, appears on both axes of the scattergram, giving rise to a spurious correlation.

Rate of Flow of Aqueous Humor in Different Species.—The rate of flow is surprisingly similar even in eyes of widely differing dimensions. Table 9 shows

TABLE 9.—*Rate of Flow of Aqueous Humor in Different Species, Expressed as Per Cent per Minute of Volume of Anterior Chamber*

	Assuming No Diffusion Through Iris	Assuming Maximal Diffusion Through Iris	Rate of Disappearance of Na^{24}
23 guinea pigs	1.7	1.4	1.6 *
08 American [2c] albino rabbits	1.3	1.1	1.1 [2c]
23 Swedish † albino rabbits	1.1	0.9	...
14 white leghorn [5] hens	2.0	1.7	...
15 cats	1.4 ‡	...	1.4
10 human eyes	1.1 [7]	1.0 §	...

* Wilde, W. S.; Scholz, R. O., and Cowie, D. B.: Am. J. Ophth. **30**: 1516 (Dec.) 1947.
† Bárány, E.: Unpublished data.
‡ Calculated by me from sodium values obtained by Davson and others,[6] assuming the volume of the anterior chamber in cats to be 1 ml. and the iris to be impermeable to sodium.
§ Calculated by me from Goldmann's data.[7]

the present information on this point. The values for the rabbit, guinea pig, and chicken have been obtained with the same method, and on unanesthetized animals. It is interesting to note that the rate of turnover of sodium is about equal to the rate of flow of aqueous in the guinea pig, as well as in the rabbit. This shows the iris vessels to be practically impermeable to sodium in both species. The same seems to be true for the anesthetized cat, in which Davson [6] and co-workers have found that sodium left the anterior chamber at the same rate as inulin. Their turnover rates for sodium thus give a measure of rate of flow in the eyes of this animal, too, and it turns out to be very similar to that in much smaller eyes. Goldmann's [7] values in human eyes were obtained with his fluorescein method,

5. Footnote deleted.

6. Davson, H.; Duke-Elder, W. S.; Maurice, D. M.; Ross, E. J., and Woodin, A. M.: Penetration of Some Electrolytes and Non-Electrolytes into the Aqueous Humour and Vitreous Body of the Cat, J. Physiol. **108**:203 (March) 1949.

7. Goldmann, H.: Der Übertritt von Fluorescein aus dem Blut ins Kammerwasser des normalen Menschen, Experientia **5**:295 (July) 1949; Über Fluorescein in der menschlichen Vorderkammer: Das Kammerwasser-Minutenvolumen des Menschen, Ophthalmologica **119**:65 (Feb.) 1950.

which in principle is very similar to the paraaminohippuric-acid method. The very great advantage of Goldmann's method is its suitability for use on human subjects and the fact that flow can be determined in a single eye. One inherent advantage that does not seem to be fully utilized at present is that several points on the time-concentration curve of one eye can be obtained. Thus, it should be possible, as Goldmann points out, to determine the constants pertaining to the posterior chamber as well. At the present stage, Goldmann, in fact, uses the same approximations as Bárány and Kinsey: He starts out with a two-chamber model but has to approximate to the equivalent of a one-chamber model when evaluating his data.

COMMENT

The present experiments do not support the view that the ascorbic-acid-dependent dye-transport mechanism of Friedenwald and collaborators is responsible for the production of the bulk of the aqueous. This is no reason that it should not be physiologically important. The high constancy of aqueous flow suggests an important nutritive function, and it seems reasonable that the redox pump is actively secreting one or several constituents of the aqueous which are necessary for the avascular tissues of the anterior part of the eye. For all we know, ascorbic acid might be one such constituent.

SUMMARY

The rate of flow of aqueous humor has been determined in unanesthetized guinea pigs supplied with widely different amounts of ascorbic acid. Except in deeply scorbutic animals, there was no difference in rate of flow despite large differences in aqueous ascorbic acid. Rate of flow was about 1.7% per minute of the volume of the anterior chamber in normal guinea pigs. In animals with marked scurvy, the rate was about 1.4% per minute.

Attention is drawn to the similarity in percentage rate of flow in different species.

Mrs. I. Gertz, Miss G. Thelin, and Miss I. L. Dalhagen gave technical assistance in this study.

New Instruments

A COMBINATION SQUARE PRISM AND LOLLIPOP HOLDER

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BROOKLYN

In a standard square plastic or glass prism, averaging about 38 mm. square, two small holes were drilled, each about 5 mm. in length and 2 mm. wide, one into the base and the other in one side of the prism. The hole in the base of the prism was drilled perpendicularly at a point midway of this rectangular surface. The hole in the side of the prism was drilled perpendicularly at a point midway

This illustration shows three threaded holes for the lollipop holder, though two are adequate.

between the apex and the basal edge. The holes were then tapped to permit the ready insertion of a rod about 6 inches (15 cm.) long, with one end threaded to screw readily into either of these spiraled openings. The result was a simple prism holder which could be changed readily to a base-down, base-in or base-out position. In a more recent model the holes were not threaded. One end of the rod holder was likewise not threaded but merely split vertically and the ends were spread slightly apart. Thus, the split end could be slipped into either hole and would fit firmly because of the springlike tension exerted by the separated split ends. The other end of this rod could be either left alone or expanded into a circular disk to serve selectively as an opaque cover.

The value of a prism held correctly by the patient is obvious, both as an assistance to the examiner in diagnosis and for training. The physician's hands are

Read before the Section on Ophthalmology at the Ninety-Ninth Annual Session of the American Medical Association, San Francisco, June 29, 1950.

free to operate a flashlight with one hand and a rotary prism with the other. The rod itself insures accuracy in that the prism is held correctly in either a horizontal or a vertical position. Moreover, it is much easier for the patient to handle a rod supported against the cheek than to have his two fingers cramped against the sides of the prism, which is usually tilted inaccurately before the eye.

In previous reports,[1] I presented first a single and then a multiple prism holder. Both holders were independent units with sturdy frameworks, which reduced the visible area seen through the prism. In the new, combined unit the prism itself is channeled to convert a modified square prism into a comprehensive prism holder without any obstruction whatever.

This prism has been produced in a colored, as well as a colorless, model. The advantage of the colored prism is that it facilitates questioning a child patient who sees a "colored light" and a "white light" with induced diplopia.

745 Eastern Parkway (13).

1. Krimsky, E.: A New Prism Holder, Tr. Am. Acad. Ophth. **51**:297, 1947; A Multiple Prism Holder, ibid. **54**:370, 1950.

AN ILLUMINATED MONOCULAR EYEGLASS LOUPE

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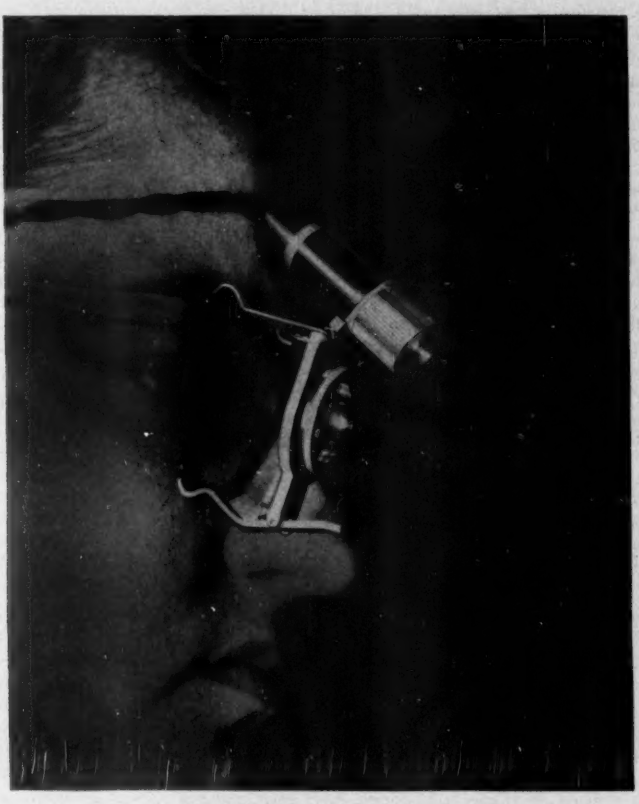
This unit consists of a light, easily maneuvered, illuminated monocular eyeglass loupe which can be made to fit firmly on any size or shape of wearing glass. The lighting unit, which is part of the instrument, serves to illuminate the field of inspection adequately.



An illuminated monocular eyeglass loupe.

The optical principle of this unit is similar to the jeweler's loupe and to a unit previously described by me.[1] In brief, its construction is as follows:

1. A magnifying biconvex (plus) lens of selective strength mounted in suitable cylindrical bed, threaded on the outside to hold an easily removable adapter ring.

Read before the Section on Ophthalmology at the Ninety-Ninth Annual Session of the American Medical Association, San Francisco, June 29, 1950.

1. Krimsky, E.: Portable Slit-Lamp and Its Clinical Value, Arch. Ophth. **30**:263-265 (Aug.) 1943.

Lenses of desired strength may be used to meet the requirements of the individual practitioner. For the present, I am depending on two strengths of lenses, + 17 D. and + 10 D. The lens housing proper is set at a convenient distance of about 1 inch from the wearer's eyeglass to increase the working distance. In the case of the + 17 D. lens the resultant working distance is 9 cm. from the eye.

2. The lighting unit is joined to the upper portion of the magnifying lens frame support and angulated downward and forward so that the projecting beam meets the lens in a common focus. Thus the magnified image is properly illuminated. The junction between the lens frame and the lighting unit has been flexibly jointed to enable ready adjustment of the light source for lenses of selective strengths.

The advantages of this improved model over ones previously reported lie in (*a*) the mechanism for clamping onto the eyeglass, (*b*) the selective interchange of lenses of desired strengths, (*c*) an improved functional design and (*d*) the ready adjustment of the light carrier to enable centralized illumination of the magnified field. The lens frame proper has two rigid, hooklike tongues which hug the wearing lens from above. To the lower part of this lens frame is attached a lever-controlled spring clasp for clamping onto the lower part of the wearer's lens frame. The illumination source for the present is a beaded miniature lamp set in a suitable housing; this is joined to a cord leading to a battery handle, which can be worn in a coat or vest pocket.

The value of such a compact illuminated magnifying system hugging either eyeglass frame is obvious. The physician's hands are then free at all times to separate the eyelids, to study the eyes more easily under proper control, to handle instruments freely and to proceed with operations on the eye when assistance is not readily available or is necessary.

A newer model at present under construction provides for the ready interchange of lenses of different strengths for operating at selective distances. Moreover, the lighting unit is being jointed where it meets the upper portion of the magnifying lens frame so that the light can always be readily centered in the magnified field for the particular type of frame or lens selected.

Registry of Interesting Cases

RECURRENT BILATERAL SCLERITIS OF MANY YEARS' DURATION WITH UNUSUAL CORNEAL INFILTRATION (FAT?)

The following case of scleritis is of interest in that the condition has persisted for 18 yr. and is accompanied with unusual corneal opacities. It has also provided an unusual opportunity to try a variety of therapeutic procedures. It appears to be clinically the same entity as that described by Uhthoff [1] as a manifestation of gout, but this etiology is not borne out either by biopsy or by therapeutic response in the present case.

A housewife aged 51 has had recurrent redness and severe pain of both eyes for the past 18 yr. These attacks occurred at first at intervals of several weeks, but during the past 10 yr. they have recurred at intervals of 8 to 14 days, affecting usually one eye at a time, each episode lasting about one week.

The patient had been well prior to the present difficulty except for rheumatic fever 22 yr. ago and sinusitis and asthma 18 yr. ago, that is, at about the time that the ocular complaints began.

The attacks of ocular inflammation have been characterized by intense redness of the conjunctival and scleral vessels, with ill-defined tenderness but no definite nodules. Approximately five years ago a glistening-white opacity occurred in the paralimbal region of the right eye, situated just anterior to Descemet's membrane, extending from 5 to 9 o'clock and separated from the limbus by a clear interval. A few vessels extended into this from the limbus. Approximately three years ago a similar opacity, also situated in the lower nasal quadrant occurred in the left eye, and since then both opacities have gradually extended toward the pupillary region (Fig.). In addition, the epithelium of both eyes has developed a faint mottling, as seen with the slit lamp, producing a mosaic appearance, with units of the order of 0.1 mm. square. There has, however, been no pyogenic infiltration or swelling of the cornea. In the past two years the episclera adjacent to the lower limbus of both eyes has been diffusely elevated by a yellowish material. Biopsy of this tissue showed considerable infiltration with polymorphonuclear leukocytes and a few giant cells. Frozen sections stained with Sudan IV (scarlet red) showed considerable fat and some birefringent crystals, which were dissolved out by means of alcohol. The murexide (Weidel) test for uric acid gave a negative result, as did colorimetric tests for cysteine.

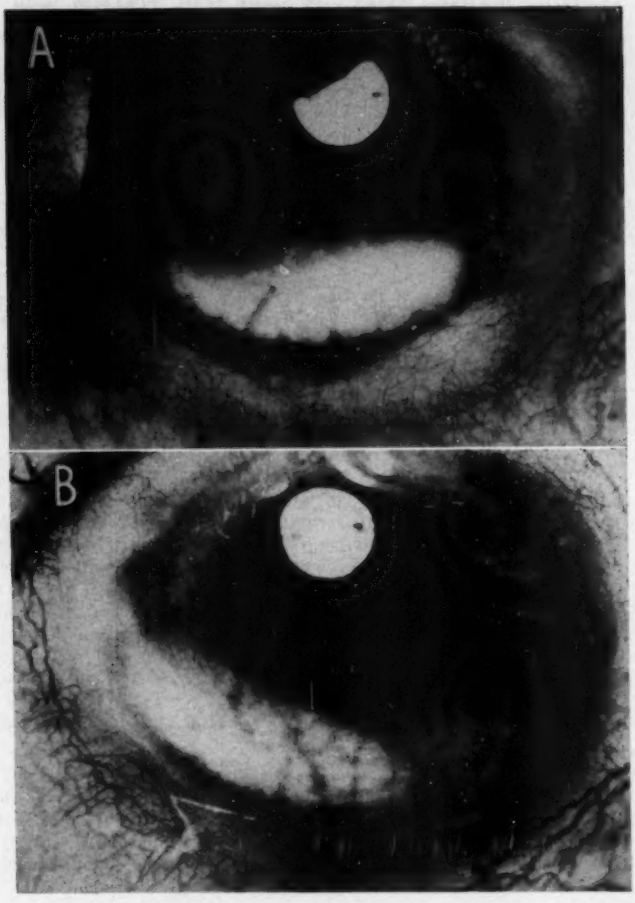
Except for the eyes, physical examination showed no significant abnormality. Roentgenograms of the chest and fingers revealed a normal condition.

Several uric-acid and cholesterol determinations on the blood have given normal values. Total fatty acids and phospholipids in the blood were normal on the one occasion on which they were tested. The sedimentation rate was somewhat elevated on one occasion, being 107 mm. in one hour (Westergren). Serum albumin was 3.5 gm., and serum globulin 4.7 gm. per 100 cc. The Wassermann and Hinton serologic tests gave negative reactions. Intradermal tests with tuberculin gave positive results only in a concentration of 1:10,000 or greater.

The following therapeutic instances were given systemically, usually on separate occasions, without benefit: colchicine U. S. P. (0.5 mg. three times a day) for two weeks; a low-fat diet for two weeks; a low-carbohydrate diet for two weeks; a low-protein diet for two weeks; diethylstilbestrol U. S. P. (1 mg. per oz. daily) for four weeks; series of Staphylococcus and Streptococcus vaccines; a tuberculin series; choline (2 gm. daily) for six weeks; inositol (1 gm. daily) for two weeks; ascorbic acid (4 gm. daily) for three months; nicotinic acid (0.6 gm. daily) for four months; thyroid U. S. P. (0.1 to 0.2 gm. daily) for two months; protamine

1. Uhthoff, W.: Doppelseitige symmetrische Degeneration der Kornea mit Ablagerungen von Harnsäure und saurem, harnsaurem Natron bei sonst normaler Beschaffenheit der Augen und gutem Allgemeinbefinden, Klin. Monatsbl. Augenh. **54**:383, 1915.

zinc insulin (15 insulin units daily) for two weeks; multiple vitamin therapy (vitamin A, 25,000 U. S. P. units; thiamine hydrochloride, 10 mg. daily) for varying lengths of time; potassium iodide; tripelennamine ("pyribenzamine") hydrochloride (50 mg. four times a day), and acetylsalicylic acid ad lib.



A, right eye, showing dense opacification of the lower part of the cornea with vascularization. The opacity was situated throughout all levels of the stroma and was separated from the limbus by a narrow, clear interval; *B*, left eye, showing same type of opacity.

There was also no evident benefit from local use of ethyl morphine hydrochloride ("dionin"), iodophthalein sodium (nosophen®) ointment, naphazoline (privine®) hydrochloride (0.1%, four times a day), cortisone (injected subconjunctivally), zinc sulfate (0.2%, three times a day), or epinephrine bitartrate (2.0%, three times a day).

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Annual Reviews

THE ORBIT

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ORBITAL tumors continue to occupy a large part of the literature on the orbit during 1950, as in the preceding year. Doherty [1] accepted Reese's classification of hemangiomas, which divides them into (1) capillary, (2) cavernous, (3) angioblastic, or hypertrophic, and (4) racemose, or cirsoid. Doherty describes the angioblastic type as a solid mass of endothelial cells with no patent vessels or a minimum of very small channels. The rapid proliferation of embryonic endothelium eliminates the lumen in hemangiomas of this type. The racemose type is composed of a pulsating mass of dilated, thickened, tortuous blood vessels and proliferating capillaries. He points out that the angioblastic and racemose types of hemangiomas usually occur in adults and have a relatively sudden onset. Because of the constant arteriolar pulsation, there may be erosion and invasion of the surrounding structures, leading often to fatal hemorrhage.

Doherty emphasizes Reese's teaching that hemangiomas, except for the racemose type, do not show progressive, unlimited growth, but tend to reach a size and become stationary, with full-bodied development, and that this should be borne in mind when one is considering radical, and possibly disfiguring, surgery in the young. There may be spontaneous regression due to thrombotic occlusion. Simple hemangioma having no capsule tends to infiltrate widely in the orbit, menacing the integrity of all the orbital structures, making removal difficult, and often followed by severe hemorrhage and sensory or motor disturbances. He believes that the prognosis is good if the condition is seen and diagnosed early.

Doherty is of the opinion that the therapy of hemangioma varies with its position in the orbit. When it is in the anterior part of the orbit, he considers injections or incision adequate. When injections are used, ligations should always be carried out first to prevent involvement of the cavernous sinus. He is of the opinion that ligations alone are not enough. When the hemangioma is in the middle third of the orbit, a transconjunctival or Krönlein approach is usually best, but when it is in the posterior third only a transfrontal approach gives adequate exposure for complete removal, with least damage to other structures.

Arteriography continues to play an important role in the diagnosis of orbital tumors. Röttgen [2] reports a case of retrobulbar angioendothelioma with pulsating exophthalmos which was outlined by angiography. An additional finding was papilledema of 4 D. Compression of the external carotid artery on the same side caused cessation of the pulsation and diminished proptosis, while compression of the opposite jugular vein produced aggravation of the symptoms. The tumor was removed by the transfrontal route, which he also feels is the best approach, since many angiomas have an intracranial extension.

1. Doherty, W. B.: Hemangioma, Am. J. Ophth. **33**:1111-1117, 1950.

2. Röttgen, P.: Retrobulbar Angioendothelioma with Pulsating Exophthalmos, Klin. Monatsbl. Augenh. **116**:256-262, 1950.

Less than four cases of neuroblastoma of the adrenal cortex (Hutchinson's syndrome) are recorded yearly. Cox [3] finds that the orbit is invariably involved in the metastasis, and frequently it is the eye which gives the first noticeable indication of the disease. In the Pepper type, the mesenteric lymph nodes and the liver are predominantly involved; the average age of the patients is 6 mo., and the duration of the disease is usually only two to five months before death intervenes. In the Hutchinson type the skull and the periorbital area are particularly affected. The original tumor may resemble the ganglioneuroma, and the metastases have a typical neuroblastomatous formation. The average age of patients with this type is 3 yr. 10 mo., and the duration of the disease before death is longer than that of the Pepper type. Usually no abdominal tumor is palpable early in the disease. Clinically, anemia is a striking feature in all cases, and it is that of a profound secondary type. Cox finds the prominent clinical characteristics of tumor of the adrenal cortex to be hirsutism, precocious puberty, and, frequently, increased blood pressure.

Harbert and Tabor [4] find that Ewing's tumor of the orbit involves the bone arising from capillary endothelium and presents much the same picture clinically and structurally as do metastatic adrenal tumors (adrenoblastoma). They believe that the skull is rarely involved (in 5% of cases), and apparently their two cases are the first to be reported with orbital involvement. They describe Ewing's tumor as a diffuse endothelioma, or endothelial myeloma, which is characterized by its marked sensitivity to radiation.

The diagnosis of sarcoidosis of the orbit is usually made by correlating the clinical features—increased eosinophil count, increased sedimentation rate, pulmonary changes, exclusion of tuberculosis and other infectious diseases, and a chronic benign course—with the microscopic appearance of the tissue removed for biopsy or at operation. Two reports of this disease were made during the past year (Bodian and Lasky [5]; Rider and Dodson [6]).

Plasmacytic tumors of the bone marrow occur frequently. Extramedullary plasmacyte tumors are unusual, and most have involved the mucous membranes. The commonest site of occurrence is the air passages, and the next commonest is the conjunctiva. Walsh described the first case of this tumor involving the orbit, and McEvoy,[7] and Geréb and Könyves-Kolonics [8] present additional cases. The tumor produces a protein in the urine which must be differentiated from albumin by the Bence Jones test. If this protein is found, the diagnosis is conclusive; however, its presence is intermittent. Bone-marrow biopsy and x-ray examination of the skull are additional diagnostic aids.

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3. Cox, R. A.: Proptosis Due to Neuroblastoma of the Adrenal Cortex (Hutchinson's Syndrome), Digest Ophth. & Otolaryng. **12**:437-441, 1950.

4. Harbert, F., and Tabor, G. L., Jr.: Ewing's Tumor of the Orbit, Am. J. Ophth. **33**:1219-1224, 1950.

5. Bodian, M., and Lasky, M. A.: Sarcoidosis of the Orbit, Am. J. Ophth. **33**:343-353, 1950.

6. Rider, J. A., and Dodson, J. W.: Sarcoidosis: Report of Case Manifested by Retrobulbar Mass, Proptosis, Destruction of Orbit and Infiltration of Paranasal Sinuses, Am. J. Ophth. **33**:117-120, 1950.

7. McEvoy, J. J.: Plasma Cell Myeloma of the Orbit: Report of a Case, Arch. Ophth. **44**:163-165 (July) 1950.

8. Geréb, T., and Könyves-Kolonics, L.: Intraorbital Localization of Multiple Myeloma, Orvosi hetil. **91**:282-284, 1950.

Craig and Gogela,[9] in reporting three cases of meningioma of the optic foramen as a cause of slowly progressive blindness, state that intraorbital meningiomas which arise from the meninges within the optic foramen are rare, grow very slowly, tend to be bilateral and exhibit the solitary symptom of progressive loss of vision. The patients complained only of progressive visual loss over many years. Operations disclosed small, cufflike tumors encircling and compressing the optic nerves, and these were removed, without resulting in improvement of vision. Such meningiomas arise from clusters of arachnoidal cells which normally occur along the intraorbital course of the optic nerve. Why there is a predilection for the location in the optic foramen or why these tumors are commonly bilateral is difficult to explain. Early recognition of the possibility of such a lesion in a case of progressive loss of vision without other symptoms might lead to the control of the process, and even to some improvement in, or restoration of, vision.

Blatt and Athanasiu[10] also stress the importance of a careful roentgenographic exploration of the optic canal in all doubtful affections of the optic nerve. They report a case of osteomas of the left sphenoid and left frontal bone, causing displacement of the eyeball and complete atrophy of the left optic nerve. After slow progression in the course of five years, the patient presented an extremely narrowed, flattened, and irregularly zigzag-shaped optic canal.

Neurinoma of the orbit was discovered by Standal[11] to occur 8 times among 32 primary orbital tumors seen in 68 consecutive cases of monocular exophthalmos. Two of these neurinomas were found to be malignant and were treated by exenteration. Most of the patients were young (2 to 18 yr. of age), and none had Recklinghausen's neurofibromatosis. Six patients were treated with good results by local excision and x-ray therapy.

Primary tumors of the optic nerve are gliomas and meningiomas, and Horns[12] believes that x-ray visualization of the optic canals is of aid in their detection. He feels that if x-ray examination shows the tumor has extended through the optic foramen, operation by the transfrontal approach should be done. If the globe must be excised, this should be done after complete healing has taken place. X-ray therapy is of little value. He finds that gliomas usually occur during the first decade of life and are usually intraorbital, but may have extended into the intracranial portion of the optic nerve, or even the chiasm, before removal. Meningiomas of the optic nerve sheath, like meningioma elsewhere, tend to occur later in life, and symptoms usually develop after the first decade of life. More than 50% occur after the age of 30. Meningiomas arising intraorbitally are rare, but they frequently extend into the orbit.

Another interesting aspect of the cause of blindness in the elderly is that of the sphenoidal fissure-optic canal syndrome presented by Tassman.[13] He states that a

9. Craig, W. M., and Gogela, L. J.: Meningioma of the Optic Foramen as a Cause of Slowly Progressive Blindness, J. Neurosurg. **7**:44-48, 1950.

10. Blatt, N., and Athanasiu, M.: Typical Deformity of the Optic Canal in Osseous Tumors of the Sphenoid, Ophthalmologica **119**:137-145, 1950.

11. Standal, B.: Neurinoma of the Orbit, Acta ophth. **28**:49-70, 1950.

12. Horns, R. C.: Primary Tumors of the Optic Nerve, Minnesota Med. **33**:241-243, 1950.

13. Tassman, I. S.: Sphenoidal Fissure-Optic Canal Syndrome: Presentation of 4 Cases, Arch. Ophth. **44**:753-756 (Nov.) 1950.

pathologic process which involves the bones at the apex of the orbit may produce pressure on the structures which pass through the sphenoidal (superior orbital) fissure and the optic canal as they enter the orbit. Syphilis and inflammation of the bones of the orbit, arising in the nasal sinuses especially, may produce ocular impairment, but perhaps a more important, and not infrequent, cause is a malignant process involving the sphenoid bones secondarily, by extension from a primary seat in the paranasal sinuses. Ptosis and ophthalmoplegia are the most important and earliest manifestations of this syndrome and result from the pressure on the structures which enter the orbit through the sphenoidal fissure. Various degrees of visual impairment result from pressure on the optic nerve as it enters the optic canal. The latter is closely related medially to the sphenoid sinus, and sometimes to a posterior ethmoid air cell. The complete sphenoidal fissure-optic canal syndrome therefore indicates involvement primarily of the second, third, fourth, and sixth cranial nerves, the ophthalmic division of the fifth cranial nerve, sympathetic fibers, and the ophthalmic artery and vein. When the syndrome is incomplete, the optic nerve or some of the other structures are spared. The patient is generally over 60, and recurring epistaxis with nasal symptoms usually are complained of early.

RETROBULBAR INJECTIONS

Leopold and associates [14] find questionable value in retrobulbar injection of cortisone in cases of retrobulbar neuritis, posterior uveitis, and macular degeneration. After each retrobulbar injection of cortisone there was noticeable edema of the lids and conjunctiva. One patient had severe pain behind and over the eye immediately after the injection. There were limitation of motion of the extraocular muscles and redness of the lids, together with edema. The eye had the appearance of a retrobulbar abscess. Usually the edema and reaction appear in 48 to 96 hr. and subside more rapidly, with ice compresses. No influence was found on the eosinophil count, the blood sugar or the blood pressure after retrobulbar or subconjunctival injections of cortisone.

Parrish and associates [15] suggested that retrobulbar injections of 150 turbidity-reducing units of hyaluronidase in 2 cc. or more of procaine-epinephrine solution, repeated daily or every other day, might prevent subsequent irreversible fibrotic changes in the orbit in early cases of thyrotropic exophthalmos. They found that this solution injected beneath the conjunctiva reduces the chemosis, so that the lids can be closed. Kadin,[16] however, using 250 viscosity units of hyaluronidase (alidase®) dissolved in 3 cc. of 2% procaine hydrochloride and injected—1 cc. retrobulbarly, 1 cc. into the conjunctival bleb and 1 cc. into the lower eyelid—found that after 20 min. there was a definite increase in the amount of swelling and proptosis. At 24 hr. the edema and proptosis were still increased, and not until 48 hr. had elapsed did the edema and proptosis return to its preinjection levels. Kadin concluded that in this case hyaluronidase was of no value in treatment of thyrotropic exophthalmos.

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14. Leopold, I. H.; Purnell, J. E.; Cannon, E. J.; Steinmetz, C. G., and McDonald, P. R.: Local and Systemic Cortisone in Ocular Disease, Am. J. Ophth. **34**:361-371, 1950.

15. Parrish, R. G.; Eason, E., and Karp, M.: General Anesthesia for Ophthalmic Surgery, Am. J. Ophth. **33**:883-889, 1950.

16. Kadin, M.: Hyaluronidase in Thyrotropic Exophthalmos, Am. J. Ophth. **33**: 962-964, 1950.

Lebensohn [17] finds hyaluronidase almost nontoxic and well tolerated subconjunctivally, retrobulbarly, subcutaneously, and intramuscularly. When injected into the cornea, vitreous, or anterior chamber, it causes severe reactions and permanent tissue changes. He advocates its use in ophthalmology primarily as an adjuvant to local anesthetics. The tension-reducing effect in normal and in glaucomatous eyes of retrobulbar injections of procaine is greatly enhanced by the addition of hyaluronidase, and Venco [18] believes that it increases the safety of the intracapsular extraction of cataract.

Lebensohn finds that among the agents that inhibit hyaluronidase are morphine, salicylates, antihistaminic drugs, and the steroid hormones activated by corticotrophin (ACTH). Glaucoma has not been noted as a complication of their extensive or intensive use. He believes that the tendency of subconjunctival injections of hyaluronidase to clear the cornea, though temporary, may occasionally permit a better study of the fundus. He does not find that its local administration increases the absorption of drugs through the cornea. Postoperative edema is less after muscle operations and enucleations when hyaluronidase is used with local anesthesia retrobulbarly.

Parrish and associates [15] suggested hyaluronidase as a retrobulbar injection in cases of postoperative persistent loss of the anterior chamber, either before or at the time air is injected, since it greatly increases the depth of the normal chamber. This suggestion was not substantiated by clinical trial.

Polan [19] uses a retrobulbar injection of 1 cc. of ethyl alcohol after 1 cc. of 2% procaine hydrochloride is injected into the muscle cone in cases of intractable ocular pain. If the pain is not relieved, it may be repeated two to three days later. He finds that the muscular paresis usually disappears in a month or two.

THYROTOXIC AND THYROTROPIC EXOPHTHALMOS

Investigations have continued during the past year in an attempt to elucidate the metabolic changes leading up to the development of thyrotropic exophthalmos. Allegranza and Fiocca [20] gave 0.5 gm. of methylthiouracil orally each day for 30 days to 30 female adult guinea pigs. The animals showed more or less pronounced bilateral exophthalmos, but histologic study of the hypothalamus failed to reveal any changes from the normal. Ludwig, Boas, and Soffer [21] produced exophthalmos in three groups of guinea pigs treated, respectively, with thyrotropic hormone alone, thyroidectomy alone, and thyroidectomy plus thyrotropic hormone. The most pronounced exophthalmos occurred in guinea pigs with simple thyroidectomy. The pathologic changes in the orbits of these animals consisted in an accumulation of ground substance in the connective tissue, mostly hyaluronic acid, with subsequent increase in water content.

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17. Lebensohn, J. E.: Hyaluronidase in Ocular Surgery and Therapy, Am. J. Ophth. **33**:865-870, 1950.

18. Venco, L.: Hyaluronidase in Ocular Anesthesia, Rass. ital. ottal. **19**:317-325, 1950.

19. Polan, C. M.: Retrobulbar Injection of Alcohol, West Virginia M. J. **46**:299-301, 1950.

20. Allegranza, A., and Fiocca, S.: The Hypothalamus of Guinea-Pigs Treated with Methylthiouracil, Biol. latina **2**:462-478, 1950.

21. Ludwig, A. W.; Boas, N. F., and Soffer, L. J.: Role of Mucopolysaccharides in Pathogenesis of Experimental Exophthalmos, Proc. Soc. Exper. Biol. & Med. **73**:137-140, 1950.

The therapy of thyrotropic exophthalmos seems, from the tone of the literature, to be swinging toward x-ray irradiation of the pituitary area. Paufique, Guinet, and Papillon [22] report 12 cases of thyrotropic exophthalmos in which striking improvement followed hormonal and x-ray therapy. The hormonal treatment is based on the presumed existence of definite types of both gonadal and thyropituitary insufficiency, with defective capillary permeability. The close association of the onset of exophthalmos with abnormal permanent cessation of procreative function strongly suggests that a specific type of gonadal insufficiency is an important factor in the causation of the thyrotropic exophthalmos.

Treatment with large doses of estrogens or testosterones is therefore strongly advised. That a specific thyropituitary insufficiency is also a causative factor is demonstrated in the clinical and laboratory tests made.

The nonelastic character of the orbital tissues, in contrast with the more elastic consistency noted in cases of thyrotoxic exophthalmos, suggests to the authors the presence of mucin, which is deposited throughout the body in some types of hypothyroidism. The authors advise appropriate treatment of the hypothyroidism with thyroxin, administered with parasympathetic-activating drugs, preferably neostigmine.

The x-ray therapy advised is of the semipenetrating type and is preferably given at the same time as the hormonal treatment. The total dose suggested is between 1,800 and 5,700 r, given in one to three weeks, and the rays are directed both to the orbit and to the pituitary gland. Orbitohypophysial roentgen therapy, in a dose of 150 to 200 r per session, repeated up to five times a week, for a total dose of from 1,800 to 5,000 r, gave good results in seven of eight cases.

Chandler [23] finds, however, that irradiation of the orbits or of the pituitary area has been given with equivocal results in cases of thyrotropic exophthalmos. He suggests that in the cases with congestion thyroid extract be pushed to the point of mild thyrotoxic symptoms. Sometimes the thyroid is combined with iodine.

In the review of the literature on the orbit for 1949,[24] it was anticipated that a large body of new data would be published in the next year referable to the use of corticotrophin (ACTH) and cortisone in investigations of thyrotropic exophthalmos. The reports, however, are few, and on the whole disappointing. Hill and associates [25] treated four patients with thyrotropic exophthalmos with corticotrophin. All patients had relief of pain, lacrimation, and chemosis to some extent. Three showed minimal decrease in measurable exophthalmos. After withdrawal of corticotrophin, one showed exacerbation of exophthalmos, and one maintained symptomatic improvement. Salassa [26] treated one patient with corticotrophin and

22. Paufique, L.; Guinet, P., and Papillon, J.: Treatment of Edematous Exophthalmos Without Hyperthyroidism, Ann. ocul. **183**:449-482, 1950.

23. Chandler, P. A.: Ocular Manifestations of Thyroid Disease, Texas State J. Med. **46**:801-802, 1950.

24. Fralick, F. B.: The Orbit: Review of the Literature, Arch. Ophth. **44**:437-453 (Sept.) 1950.

25. Hill, S. R., Jr.; Reiss, R. S.; Forsham, P. H., and Thorn, G. W.: Effect of Adrenocorticotropin and Cortisone on Thyroid Function: Thyroid-Adrenocortical Interrelationships, J. Clin. Endocrinol. **10**:1375-1400, 1950.

26. Salassa, R. M.: Effects of Cortisone and ACTH in Certain Endocrine Conditions, Proc. Staff Meet., Mayo Clin. **25**:497-498, 1950.

two with cortisone. He noted no significant change in the exophthalmos. The periorbital edema and the conjunctival chemosis were unchanged in two patients and were made worse in the third.

In an attempt to determine the degree of asymmetry of exophthalmos in cases of thyrotoxicosis, Drescher and Benedict [27] measured the eyes of 100 normal subjects and found that the asymmetry fell within a range of 10 to 24 mm., with a mean of 17.3 mm. The maximal normal asymmetry of the eyes was 2.0 mm. They found that an asymmetry of exophthalmos of the two eyes in excess of 7 mm. is uncommon in cases of exophthalmic goiter.

VASCULAR LESIONS INVOLVING THE ORBIT

Walander [28] has collected 800 cases of aneurysm of the carotid artery and cavernous sinus from the literature. Analysis of these cases revealed that the pulsating exophthalmos was most frequently unilateral. The aneurysm affected the abducens nerve in 62%, the oculomotor nerve in 28%, and the trochlear nerve in 10%. Spontaneous healing occurred in 5.6% of 322 cases. He found, however, that there is considerable danger of recurrence after spontaneous healing or surgical treatment by ligature of the carotid artery.

Levitt [29] finds that aneurysm of the internal carotid artery may produce a syndrome consisting of ophthalmoplegia and primary optic nerve atrophy, simulating the sphenoidal fissure-optic canal syndrome [13] or neoplasm at the base of the skull. A roentgenogram of the skull may show erosion of the optic canal and adjacent border of the orbital plate, erosion of the sphenoidal ridge and/or destruction of the dorsum sellae and the posterior clinoid processes. Not in all cases is the syndrome complete. He believes that transfrontal craniotomy is the approach of choice. Severe ocular pain and frontal headaches in the presence of ophthalmoplegia should make one suspect the presence of aneurysm. Bony changes found in the roentgenogram and arteriogram enable one definitely to establish the diagnosis in the majority of cases.

Taptas [30] propounds his theories on the etiology and pathogenesis of pulsating exophthalmos after reviewing the present theories of arterial aneurysm and a communication between the carotid artery and the cavernous sinus. He states that autopsy or arteriography shows the occurrence of an arteriovenous anastomosis in no more than 48% of cases. The mechanism of the pulsating exophthalmos, he thinks, is explained by a neurovascular disturbance, a vasodilative disorder. This pathologic vasodilatation may affect the arterial and venous elements. The occurrence of arteriovenous aneurysm is assumed to be due to the dilation of previously existing direct communications. He is of the opinion that these abnormal vasomotor responses occur on a basis of endocrine disorders (impaired hypophysio-

27. Drescher, E. P., and Benedict, W. L.: Asymmetric Exophthalmos, Arch. Ophth. **44**:109-128 (July) 1950.

28. Walander, A.: Carotid-Cavernous Aneurysm Simulating Acute Ethmoiditis, Acta otolaryng. **38**:246-249, 1950.

29. Levitt, J. M.: Aneurysm of the Internal Carotid Artery Simulating Tumor, Arch. Ophth. **43**:500-502 (March) 1950.

30. Taptas, J. N.: Etiology and Pathogenesis of Exophthalmos of Vascular Origin, Termed Pulsating Exophthalmos, Arch. opht. **10**:22-50, 1950.

corticoadrenal function) and may be set off by trauma or a toxic bacterial infection. The pulsating exophthalmos he considers to be only an anatomoclinical form of the neurovascular disease.

Pulsating exophthalmos may be produced by conditions other than those mentioned above, as has been pointed out by Contino.[31] He classifies pulsating exophthalmos as follows:

Pulsating exophthalmos
- (*a*) True, due to intracavernous aneurysm
  - Due to aneurysm between internal carotid artery and internal jugular vein
  - Due to aneurysm of ophthalmic artery
  - Due to cavernous-sinus thrombosis
- (*b*) Pseudo, due to neoplasm
  - Due to intraorbital pulsating neoplasm
  - Due to orbital hemorrhage

The clinical findings assisting in the diagnosis of orbital hemorrhage as the cause of pseudopulsating exophthalmos he finds to be hemorrhagic chemosis of the conjunctiva, irreducibility of the exophthalmos, ophthalmoscopic ischemia of the papillary vessels, changes in the radial pulse, and spontaneous cure, with absorption of the hemorrhage.

MISCELLANEOUS CONDITIONS AFFECTING THE ORBIT

That bilateral orbital granuloma may simulate a neoplasm is well exemplified by the report of Hogan and Dickson.[32] After slight injury, severe progressive exophthalmos with edema of the lids and ptosis developed in a 2-mo.-old girl. Orbital exenteration revealed a neuromyxofibroma. One year later exophthalmos developed in the other orbit, coincidentally with sinus infection. Transfrontal craniotomy was followed by recession of the exophthalmos. Only inflammatory tissue was found in the orbit on biopsy, suggesting thyrotropic exophthalmos coincidental with a true tumor.

Bentivoglio[33] reports little effect from the administration of streptomycin and paraaminosalicylic acid in a case of tuberculous granuloma of the orbit, which he considered exogenous and traumatic in origin. Orbital tuberculosis, may however, arise from the periosteum of the orbital margin, from the lacrimal glands or sac, by extension from the globe or optic nerve sheath, and from the nasal accessory sinuses.

As an example that the body may be intolerant to any substance with which it comes in contact, Forster and Dickey[34] report on sensitivity to a gold-ball orbital implant. The patient had an enucleation and was fitted with gold-ball implant in 1939, and in 1944 a purulent discharge from the left orbit developed. After two

31. Contino, F.: False Pulsating Exophthalmos Due to Orbital Hemorrhage, Rass. ital. ottal. **19**:196-208, 1950.

32. Hogan, M. J., and Dickson, O. C.: Bilateral Orbital Granuloma Simulating a Neoplasm, Tr. Am. Acad. Ophth., pp. 573-577, May-June, 1950.

33. Bentivoglio, M.: A Rare Case of Tuberculous Granuloma of the Orbit, Rass. ital. ottal. **19**:357-369, 1950.

34. Forster, H. W., Jr., and Dickey, R. F.: Sensitivity to Gold Ball Orbital Implant; Eczematous Contact Dermatitis Due to 14-Karat Gold: Report of a Case, Arch. Ophth. **44**:170-171 (July) 1950.

and a half years of treatment, without success, the question of sensitivity to the gold-ball implant was raised. It was found that the gold-ball had 60% fine gold, 35% copper, and 5% silver and tin. Patch tests gave negative reactions for all the metals but gold, and the gold ball was removed, with cure.

A warning to those administering x-ray therapy about the eye that irreparable damage may be inflicted to the eye and its surroundings by its indiscriminate use is given by Knüpffer.[35] He reports four cases, in two of which deep, irreparable necrosis of the bone followed x-ray treatment for carcinoma of the skin. In a third case a tumor of the hypophysis was treated with high doses of x-rays; the results of this was an affection of the retina resembling retinitis circinata.

A troublesome condition encountered by all ophthalmologists is that of chronic inflammation of the orbital socket after enucleation. Macivor[36] studied 120 cases of chronic and acute recurrent inflammation of the orbital socket. He found sodium sulfacetimide to be the most useful drug in the treatment of these conditions. Streptomycin is indicated in infections caused by Bacillus pyocyaneus, but after local therapy for five days the organisms tend to become resistant. Aureomycin hydrochloride is particularly useful in the treatment of Proteus vulgaris and Bacillus coli infections.

ORBITAL IMPLANTS

A striking feature of the literature on orbital implants during 1950 is the sharp swing away from the integrated implant and the development of various types of buried implants. Troutman[37] attempts to maintain partial integration by means of a magnetic field between the implant and the prosthesis. The implant is made of plastic and is entirely covered with Tenon's capsule and the conjunctiva. It encloses the "alnico" magnet. The neck of the implant is countersunk and contains a double band, 3 mm. wide, of tantalum mesh, to which the muscles are attached. The anterior surface of the implant is flat.

Allen and Allen[38] carry the flat anterior surface throughout the tunneled hemispherical plastic implant, so that this large flat surface is in contact with the flat posterior surface of the prosthesis. The merits claimed for the flat-surfaced buried implant are the security from infection and the reduced danger of extrusion. The implant gives up to 15 to 20 degrees of rotation from the primary position. The muscles should be separated from their fascial sheaths for 15 mm. The conjunctiva and Tenon's capsule are closed without tension. Although the movements are not as great as with the pegged implant, short and immediate movements are approximately as good.

Landegger[39] has developed a buried implant which utilizes all six extrinsic muscles. The advantage claimed for this implant is that the oblique muscles form a

35. Knüpffer, N.: Rare X-Ray Damage of the Eye and Its Surroundings, Klin. Monatsbl. Augenh. **116**:66-77, 1950.

36. Macivor, J.: The Management of Chronic Inflammation of the Orbital Socket, Am. J. Ophth. **33**:1573-1586, 1950.

37. Troutman, R. C.: The Magnetic Implant, Arch. Ophth. **43**:1123-1124 (June) 1950.

38. Allen, J. H., and Allen, L.: A Buried Muscle Cone Implant: I. Development of a Tunneled Hemispherical Type, Arch. Ophth. **43**:879-890 (May) 1950.

39. Landegger, G. P.: A New Orbital Implant Which Uses the Six Extrinsic Muscles, Arch. Ophth. **44**:729-730 (Nov.) 1950.

sling around it and push it slightly forward. The implant thus becomes more permanently placed in the center of the orbit, and the lower lid stays in its proper position and has no tendency to sag.

McDowell [40] has attempted a compromise between a buried and a pegged implant by devising a removable and permanent orbital implant. The socket is prepared for the implant by creating a skin-lined, semicircular tunnel in its posterior wall. The tunnel is vertically placed, with its lower mouth just back of the tarsal border of the lower lid and its upper mouth about 3 mm. below the apex of the superior sulcus of the socket. The midsection of the tunnel is posterior to the fused, or sutured, ends of the extraocular muscles. The implant is a semicircular gold band with a straight gold band bridging vertically across the two ends; the straight band is hinged to one end of the curved piece and snaps into the other end. The straight band has a square hole (3 by 3 mm.) in its midsection for reception of a similar square peg, which is fused onto the back of the prosthetic eye. The advantage claimed for this implant is that it lies in a skin-lined tunnel and thus is not a buried implant, which is likely to be extruded. Since the implant can be removed and the tunnel washed out from time to time, it is thought that the socket will remain cleaner than one containing an irremovable spherical implant surrounded by raw surfaces that discharge.

SURGERY OF THE ORBIT

One of the most refreshing reports on orbital surgery during the past year is that by DeVoe.[41] It is seldom that an author has the courage to write a second article stating that a previous article by him presenting a new operative procedure should be disregarded, since the end results were unsatisfactory. Usually physicians are left to find this out for themselves. DeVoe gives a follow-up report on the use of glass wool in the restoration of fractures of the orbital floor. He now reports that the glass wool was extruded in nine of 19 cases and concludes that in its present form it is not satisfactory for use in this manner. He believes recourse should be made to autogenous tissues, such as bone and cartilage. An excellent article by Converse and Smith [42] on reconstruction of the orbital floor by bone grafts describes the methods used under various circumstances.

Sherman [43] has written an excellent article reviewing the literature during the past hundred years on the methods of preventing or correcting the retracted eye socket. He concludes that a sufficiently large implant at the time of enucleation or evisceration is the chief means of preventing a retracted socket. The author cannot agree with Guyton that a sphere implanted in Tenon's capsule should never be larger than 18 mm., and that 14 mm. is the optimum. Except in the case of an infant or of a

40. McDowell, F.: A New Removable and Permanent Orbital Implant, Am. J. Ophth. **33**:953-958, 1950.

41. DeVoe, A. G.: Repair of Orbital Deformities with Glass Wool, Arch. Ophth. **44**:514 (Oct.) 1950.

42. Converse, M., and Smith, B.: Reconstruction of the Floor of the Orbit by Bone Grafts, Arch. Ophth. **44**:1-21 (July) 1950.

43. Sherman, A. E.: The Retracted Eye Socket, Tr. Am. Ophth. Soc. **48**:615-635, 1950.

shrunken or badly lacerated globe, an 18-mm. implant is the usual optimum; and at times a 20-mm. sphere may be used without undue tension on the closure of Tenon's capsule.

Sherman states that the first step in correcting a retracted socket is the use of the late implant, if one is not already present. Implantation of a Wheeler grooved glass sphere in the muscle cone behind Tenon's capsule is the method of choice, since it will add an implant of larger volume than can be placed in a reopened Tenon's capsule.

If an implant is already present, additional correction can be obtained by placing an implant on the floor of the orbit. Preserved cartilage, bone, wedge-shaped acrylic, or glass wool (see warning of DeVoe [41]) will serve well for this. The author prefers acrylic because no one has demonstrated that it undergoes change or causes appreciable tissue reaction when buried. It is easily obtainable and has no porous spaces, in which blood and serum can collect. Sherman believes that implants to the upper eyelid should be avoided.

Loss of the lower cul-de-sac can often be corrected by moving the conjunctiva downward into a pocket dissected down to the orbital rim. This can be combined with the operation for late implant, thus giving greater relaxation of the conjunctiva for the new fornix. If insufficient conjunctiva is present, mucous membrane from the lower lip is much preferred to the Tiersch graft.

For exploration of the orbit, Summerskill [44] believes that, unless the preoperative investigations indicate the desirability of a specific form or orbitotomy, a transconjunctival approach combined with a free canthotomy provides a satisfactory method of exposing the orbit and dealing with any lesion found. He suggests that a free lateral canthotomy be carried out down to the orbital margin and the lateral palpebral ligament and the palpebral fascia be freed from their attachment to the periosteum. The horizontal incision in the conjunctiva is extended upward and downward, keeping a few millimeters to the bulbar side of the fornix. If the tumor is palpated in the cone, the lateral rectus is severed from the globe and the eye fully adducted and the contents of the cone exposed. If necessary, the incision in the skin may be extended along the orbital margin to permit a modified Krönlein exposure.

Larmande,[45] in writing on decompressive trephination of the optic canal, states that the optic nerve, as well as the artery by which it is fed, is frequently constricted in the region of the optic canal. In this case a rapid decompression of the affected nerve becomes imperative. Since Schloffer's first operation in 1913, trephination of the optic canal has been recommended in cases of pathologic craniosynostosis, of acute and chronic edema of the optic nerve (Bregeat [46]), of certain meningeal, gliomatous (Tönnis [47]; Bucy, Russell, and Whitsell [48]), and vascular tumors, and

44. Summerskill, W. H.: The Exploration of the Orbit, Brit. J. Ophth. **34**:292-302, 1950.

45. Larmande, A. M.: Decompressive Trephination of the Optic Canal, Afrique franç. chir., pp. 151-154, 1949.

46. Bregeat, P.: Surgical Treatment of Acute Edema of the Optic Nerve, Bull. méd. **64**:365-368, 1950.

47. Tönnis, W.: The Operative Treatment of Tumors of the Optic Nerve Which Extend Beyond the Optic Foramen, Acta neurochir. **1**:52-71, 1950.

48. Bucy, P. C.; Russell, J. R., and Whitsell, F. M.: Surgical Treatment of Tumors of the Optic Nerve: Report of a Case, Arch. Ophth. **44**:411-418 (Sept.) 1950.

of injuries of the skull. Dejean, Gros, and Cazaban [49] believe it is indicated in patients under 60 with syphilitic optic nerve atrophy if visual acuity is between 1/10 and 3/10, and even in cases of higher acuity when the condition is progressive and vision in the other eye is already lost as a result of the same process. Two routes of approach were followed: an orbital and a cranial one. By means of the orbital route, the canal may be relieved from the bottom to the top, or from within outward through the ethmoidal cells. The majority of neurosurgeons now prefer the cranial route, since it renders possible an investigation of the cisterna chiasmatica. Some operators proceed along the outside of the dura mater; others open it to a considerable extent.

CONGENITAL ANOMALIES OF ORBIT

Classification of congenital anomalies of the orbit becomes more complex with the passage of years because of the many transitional forms and the incompleteness of characteristics in each case, thus leading to the tendency to make subgroupings. According to Esteban,[50] all theories of etiological possibilities lead to the conclusion that the actual state of knowledge of such subjects as dysgenesis, dysangiogenesis and inflammations is no more than hypotheses which deal with the manifold causes capable of activating the growth of bones and the deposit of lime salts around the sutures. He discusses the roentgenologic syndrome of lordosis of the basis cranii of Bertolotti, consisting of cranio-orbital dysostosis with synostosis, cranial hypertension, deformities of the skull, exophthalmos, divergent strabismus, bilateral edema, atrophy of the optic nerve, and nystagmus. For this condition he recommends early decompressive trephination.

Decompressive trephination of the skull, followed by linear craniotomy and interpolation of a tantalum plate between the divided margins of the bones of the skull, in order to produce a marked regression of the edema of the optic disk, was reported by Heinc and Maly[51] in their case of premature cranial synostosis. Most of the articles dealing with premature cranial synostosis emphasize the necessity of early recognition and neurosurgical treatment, in order to forestall eventual catastrophe.

O'Connor and Conway [52] give the plastic procedures necessary to correct the deformities that occur in dysostosis mandibulofacialis. From the surgical point of view alone, rehabilitation of affected persons is a costly and prolonged procedure, leaving much to be desired in the end result. Jervis [53] and others have shown the hereditary patterns which these various syndromes take, leading to a tremendous sociological problem.

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49. Dejean, C.; Gros, C., and Cazaban, R.: Treatment of Syphilitic Optic Atrophy by Opening of the Optic Canal: Report of Three Cases, Montpellier méd. **37**:139-143, 1950.

50. Esteban, M.: Roentgenologic Syndrome of Lordosis of the Basis Cranii of Bertolotti, Arch. Soc. oftal. hispano-am. **10**:122-136, 1950.

51. Heinc, A., and Maly, V.: Premature Cranial Synostosi, Českoslov. oftal. **6**:8-15, 1950.

52. O'Connor, G. B., and Conway, M.: Treacher Collins Syndrome (Dysostosis Mandibulo-Facialis), Plast. & Reconstruct. Surg. **5**:419-425, 1950.

53. Jervis, G. A.: Gargoylism (Lipochondrodystrophy): A Study of 10 Cases, with Emphasis on the Formes Frustes of the Disease, Arch. Neurol. & Psychiat. **63**:681-712 (May) 1950.

PARASITIC CYSTS OF THE ORBIT

Parasitic cysts of the orbit attract little attention in this country because of their rarity. In various parts of the world they continue to be a problem for recognition and therapy. Bevacqua and Wirth [54] report an orbital cyst due to Filaria conjunctivae Addario. The cyst was opened, and there emerged a parasite 131 mm. long. It was identified as an adult female Dirofilaria conjunctivae (Addario 1885) by the striations, the form of the head, and the buccovalvar relations. It is a rare human parasite. Worthy of note was the great number of eosinophils inside the cyst, in contrast with their absence in the peripheral blood.

METHODS OF EXAMINING ORBITAL CONDITIONS

Means and Stanbury [55] have been doing clinical orbitonometry by means of an instrument for the measurement of orbital resistance. They find orbital resistance increased in the active phase of the ophthalmopathic type (thyrotropic) of Graves's disease, diminished in the inactive phase, and little changed in the ordinary (thyrotoxic) type. Cooper [56] finds increased orbital tension in some cases of Cushing's disease (pituitary basophilism).

ORBITAL TRAUMA

Martin [57] pleads for early replacement of the foundation structure after injuries to the face and jaw. He feels that a depressed orbital floor in fractures of the zygoma must be repositioned under direct view by entering the sinus by the Caldwell-Luc approach, the antrum cleared of debris, drainage provided into the nose, and the position of the zygoma maintained with packing.

LESIONS OF THE LACRIMAL GLAND AFFECTING THE ORBIT

Törnquist [58] reports a case of giant follicular lymphadenopathy in a man aged 46. After he had had generalized lymphadenoidal enlargement for two years, lymphoid infiltrations entered the conjunctival fornices, and there was invasion of the lacrimal glands and the wall of one lacrimal sac. Probably as a secondary phenomenon to the involvement of the lacrimal gland, keratoconjunctivitis sicca developed; it was successfully treated by means of canalicular diathermy. Dramatic, but temporary, regression followed x-ray therapy, and also the intravenous injection of radioactive phosphorus. After four years of the disease, the patient is still in good condition, and his body weight is almost unchanged.

201 S. Main St.

54. Bevacqua, R., and Wirth, A.: Cyst of the Orbit Due to Filaria Conjunctivae Addario, Ann. parasitol. **25**:36-41, 1950.

55. Means, J. H., and Stanbury, J. B.: Clinical Orbitonometry, Am. J. M. Sc. **220**:357-361, 1950.

56. Cooper, A. C.: The Orbital Tension in Some Cases of Cushing's Disease, Nederl. tijdschr. geneesk. **2**:1334-1336, 1950.

57. Martin, B. C.: Early Management of Face and Jaw Injuries, Ohio M. J. **46**:241-242, 1950.

58. Törnquist, R.: Ophthalmic Symptoms in Giant Follicular Lymphadenopathy, Arch. Ophth. **44**:842-853 (Dec.) 1950.

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An Evaluation of the Surgery of Congenital Cataract. Dr. Frederick C. Cordes (by invitation).

This paper will be published in full in a future issue of the Archives.

Principles of Antibiotic Therapy in Ophthalmology. Dr. Irving H. Leopold (by invitation).

New antibiotics are constantly being formed, discovered, purified, and investigated experimentally and clinically. Because of the nature of the blood-aqueous barrier, dosage schedules for new antibiotics for other tissues of the body cannot be applied automatically to infections in ocular tissues. One must be certain that the drug employed will reach an effective concentration in the infected tissue. Therefore, knowledge of the ability of each antibiotic to penetrate the intraocular tissues and fluids is essential in the control of ocular infections. The ophthalmologist must be aware also of the toxic symptoms of each drug for other tissues of the body, as well as for ocular structures.

Recent studies by Cannon, Leopold, and Nichols have demonstrated that terramycin penetrates poorly into the intraocular tissues and fluids when administered locally. The best results were obtained by subconjunctival injection of terramycin. A concentration of 5 mg. per cubic centimeter was well tolerated by the normal cornea. This concentration does not seem to inhibit epithelial regeneration in standardized corneal abrasions and is effective in controlling standard experimental corneal infections with a variety of organisms. Terramycin compares favorably with other antibiotics in the control of standard experimental infections of the rabbit cornea. Orally administered terramycin failed to produce detectable levels in the intraocular fluids and tissues of normal rabbit eyes. Intravenously administered terramycin, however, could be detected in low concentration in many of the tissues and fluids of the rabbit eye.

Data are available which suggest that experimental observation obtained on the penetration of various antibiotics into the experimental animal eye may be applied to the human eye. Recently it has been shown that chloramphenicol U. S. P. and para-aminosalicylic acid penetrate into the human eye when systemically or locally administered. The degree and rate of penetration are not as great as that found in rabbit eyes but show the same trend.

The antibiotics show similar toxicities. Practically all of them have been reported as producing hypersensitivity reactions. These are most pronounced with local application of the drug, as in the form of ointments or drops. Because of the possibility of a sensitivity reaction to any antibiotic, the ophthalmologist might avoid subsequent difficulty by using antibiotics that are less likely to be used systemically. Antibiotics such as bacitracin, polymyxin, and neomycin produce renal damage when systemically administered, and therefore, although they can be used systemically, they are not antibiotics of first choice for this purpose. In addition, neomycin has been shown occasionally to produce damage to the ear. Bacitracin is definitely effective against Gram-positive organisms; polymyxin, against many Gram-negative organisms, and neomycin, against both Gram-positive and Gram-negative organisms.

These drugs have been shown to be tolerated and effective when applied locally in the proper concentration. If the patient acquires sensitivity to any of these agents, very little is lost. If the studies of Theodore are confirmed, sodium propionate may also be considered in this category.

Antibiotics in combination may be synergistic, additive, have no influence, or interfere in their effects on one another. Combinations of such drugs as bacitracin and penicillin seem to be more effective than use of either drug alone in overcoming infection with Treponema pallidum. Various combinations of penicillin and sulfonamides have been shown to be additive in their effects. Combinations of penicillin and streptomycin have been shown to be superior to either drug alone in controlling mixtures of Gram-positive and Gram-negative organisms. However, there are combinations of antibiotics in which one antibiotic actually interferes with the action of one of the others. For example, it has been shown by Jawetz that chloramphenicol interferes with the action of penicillin. Spicer has analyzed various combinations of antibiotics and has found that aureomycin sometimes interferes with the action of penicillin or of streptomycin. Each individual strain of organisms will show variations in its reactions to various antibiotics and to combinations of antibiotics.

The ophthalmologist will be wise in not using any combination of antibiotics unless it has been definitely shown by experimental and clinical trial to be superior to a single antibiotic.

There is no doubt that bacteria acquire resistance to antibiotics. This has been shown to occur with penicillin, streptomycin, aureomycin, terramycin, chloramphenicol, neomycin, polymyxin, and bacitracin. Occasionally, organisms which were not sensitive to the antibiotic used may actually grow more rapidly in the presence of this antibiotic. This has been demonstrated for the Proteus organism in the cul-de-sac of the rabbit eye when penicillin drops were applied locally and has been demonstrated clinically with the overgrowth of Candida albicans, leading to the development of moniliasis, following the use of such agents as penicillin, aureomycin, terramycin, and chloramphenicol. Whether this is due to suppression of the normal bacterial flora, leading to a more favorable pH for the growth of the resistant organism, or to the reduction of some substance which tends to inhibit the growth of these organisms, or to a depletion of some substance from the body tissues, or to the rendering of more nutrition available for the growth of the organism by killing off competitive organisms is not known. It is possible that the organisms which are inhibited in their growth by the chosen antibiotic may have been producing a substance which was antibiotic to the organism which is now overgrowing in the presence of the chosen antibiotic.

Most ocular infections due to bacteria can be controlled by antibiotics now available, and some of the ocular infections due to viruses can also be controlled. In the selection of an antibiotic, one should be guided wherever possible by the results of bacterial cultures and sensitivity studies. Where multiple choices are available, one should use the safest, the least toxic, and the easiest to administer. Sometimes sensitivity studies are not accurate, and the final test lies with clinical trial. If clinical trial reveals that the chosen antibiotic has failed to control the infection in 24 to 48 hr., a change to another antibiotic to which the bacteria is theoretically sensitive should be made. Therapy should be continued at least 48 hr. after all clinical signs of the disease have disappeared.

The Clinical Use of Antibiotics. Dr. Perrin H. Long (by invitation).

Until recently, the choice of the antibiotic to be used in the treatment of a given infection was relatively easy. If the disease was produced by Gram-positive micro-organisms, the physician considered whether or not penicillin was indicated, whereas if a Gram-negative bacterium was the causal agent streptomycin might be used. In the last three years, after the introduction of chloramphenicol, aureomycin,

and terramycin as therapeutic agents, the choice of which is the most effective and practical antibiotic for the treatment of a given infection has become much more complex. The physician must consider which antibiotic is most effective and then weigh this against its method of use, its toxicity, and the over-all costs of its administration.

The new antibiotics—chloramphenicol, aureomycin, and terramycin—have wide ranges of activity as compared with the older agents. The three show varying degrees of activity against infections produced by both Gram-negative and Gram-positive bacteria. Each has some degree of effectiveness in certain virus infections, such as psittacosis, lymphogranuloma venereum, and primary atypical pneumonia. All three have relatively low degrees of toxicity. At present it is difficult, except in a broad manner, to make strong claims for one over the other two. About the only exception to this statement is that chloramphenicol is effective in the treatment of typhoid, whereas aureomycin and terramycin are not. Only time, and careful clinical investigation, will permit us to make specific choices with accuracy. In the meantime, each physician, when confronted with making the proper choice of an antibiotic for the treatment of a patient, must take into account the current data regarding the antibiotics and make his selection accordingly.

News and Notes

EDITED BY DR. W. L. BENEDICT

GENERAL NEWS

Annual Spring Congress, Gill Memorial Eye, Ear and Throat Hospital.—The 25th Annual Spring Congress of the Gill Memorial Eye, Ear and Throat Hospital will be held April 7 to 12, 1952, at Roanoke, Va. The following physicians will be among the guest speakers: Francis H. Adler, Hermann M. Burian, Paul A. Chandler, John M. Converse, Kenneth M. Day, John H. Dunnington, Edwin B. Dunphy, Watson Gailey, Henry M. Goodyear, Bayard T. Horton, Howard P. House, C. L. Jackson, Surgeon General Lamont Pugh, Hunter H. Romaine, Frank B. Walsh, and James C. White.

Annual deSchweinitz Lecture.—The Fourteenth Annual deSchweinitz Lecture, Sponsored by the Section on Ophthalmology, College of Physicians of Philadelphia, will be given on Thursday, Nov. 15, 1951, by Dr. Peter C. Kronfeld, of Chicago. His subject will be "Tonographic Studies of Early Glaucomas."

Need in Japan for Recent Ophthalmological Literature.—American ophthalmologists on various missions in Japan report that Japanese ophthalmologists have a real need for recent, first-rate ophthalmological literature. This need is strongest in the field of American and British ophthalmological textbooks copyrighted after 1940, as well as for complete volumes of journals of the same period.

Shortly after the close of World War II, the Lucien Howe Library of Ophthalmology acted as agent for the distribution of wartime American ophthalmological publications to various foreign centers and investigators. This library is ready to assume this role again. A fund established by Mrs. Elizabeth Procter is still available to defray costs. Donations of literature of the type described should be sent to Charles Snyder, librarian, Lucien Howe Library of Ophthalmology, 243 Charles St. Boston 14. Donors will be repaid for postal charges.

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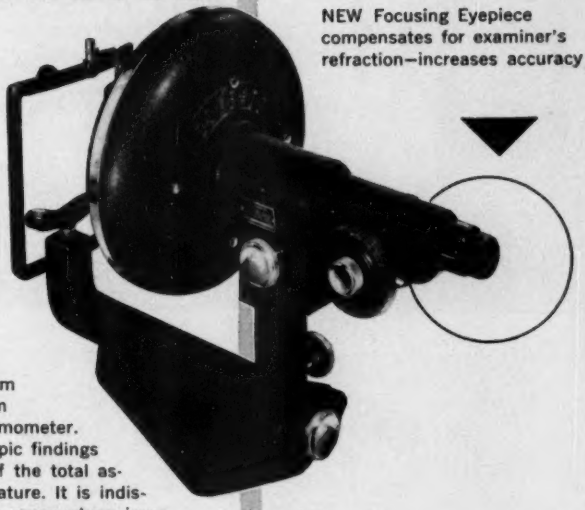

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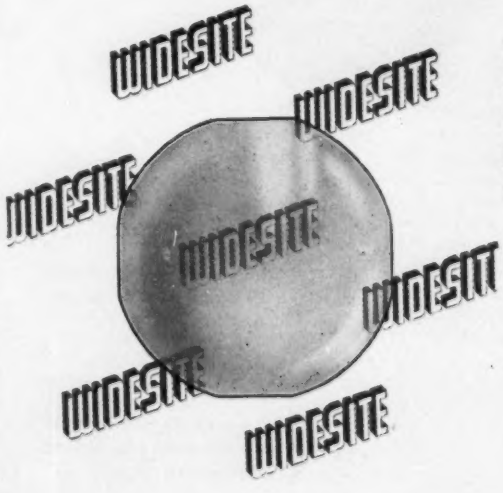

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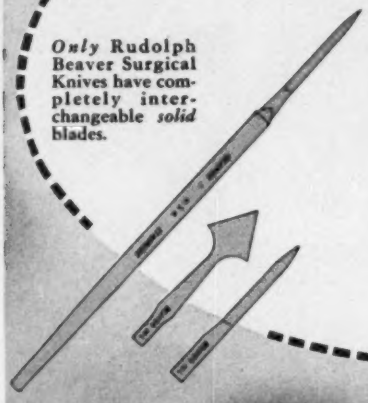

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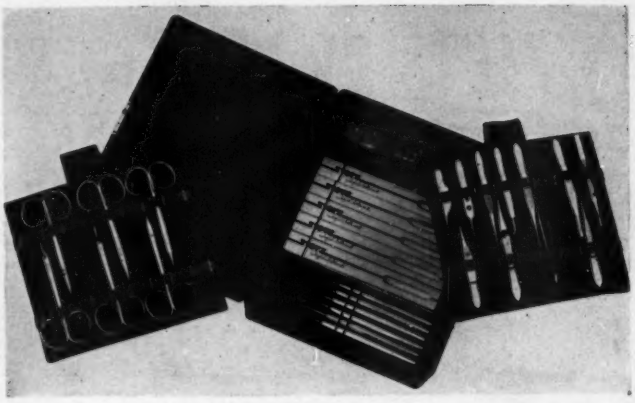

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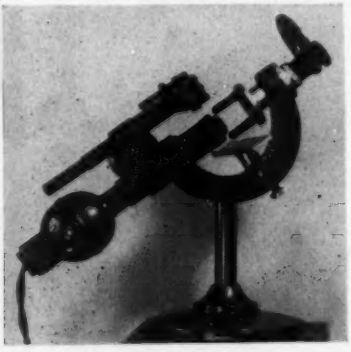

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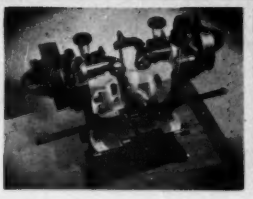
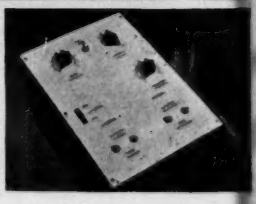
LYLE MAJOR AMBLYOSCOPE

by Clement Clarke

OF ENGLAND





The illustration depicts the oscillating mirror model in use. The automatic control unit seen fitted to the side of the Instrument Table operates the automatic flashing device and automatic and hand oscillating mirror devices.

The apparatus is designed to retain all the best features of the Moorfields Synoptophore, generally accepted as the standard equipment for Orthoptic Clinics, and to include many improvements which have been suggested by Orthoptists and Surgeons.

It is also produced in a standard form, without oscillating mirrors or automatic flashing devices. In all other respects it is identical to the instrument illustrated.

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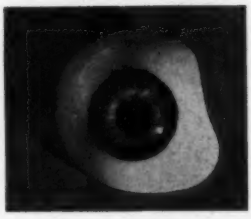

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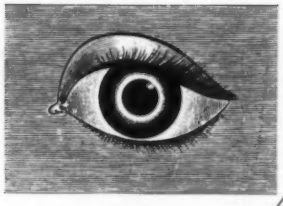

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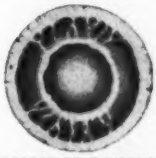
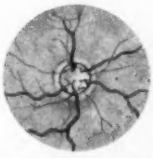
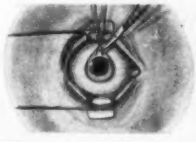


UVEITIS, POSTERIOR SYNECHIAE:
10% solution or 10% emulsion.
For freeing recently formed posterior synechiae as well as for prevention of synechiae formation in uveitis (with atropine).



GLAUCOMA (certain cases and tests):
10% solution, 10% emulsion or 2.5% solution.
For temporary reduction of intra-ocular tension and for the provocative test for angle block.



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